PB190252

AIR QUALITY CRITERIA
FOR
SULFUR OXIDES

Reproduced by the
CLEARINGHOUSE
for Federal Scientific & Technical
Information Springfield Va. 22151

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Consumer Protection and Environmental Health Service

186

AIR QUALITY CRITERIA
FOR
SULFUR OXIDES

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Consumer Protection and Environmental Health Service
National Air Pollution Control Administration
Washington, D. C.
January 1969

[illegible] 1.50

Preface

Air quality criteria tell us what science has thus far been able to meaure of the obvious as well as the insidious effects of air pollution on man and his environment. Such criteria provide the most realistic basis that we presently have for determining to what point the levels of pollution must be reduced if we are to protect the public health and welfare.

The criteria that we can issue at the present time do not tell us all that we would like to know. If all of man's previous experience in evaluating environmental hazards provides us with a guide, it is likely that improved knowledge will show that there are identifiable health and welfare hazards associated with air pollution levels that were previously thought to be innocuous. As our scientific knowledge grows, air quality criteria will have to be reviewed and, in all probability, revised. But the Congress has made it clear that we are expected, without delay, to make the most effective use of the knowledge we now have.

The Air Quality Act of 1967 requires that the Secretary of Health, Education, and Welfare ". . . from time to time, but as soon as practicable, develop and issue to the States such criteria of air quality as in his judgment may be requisite for the protection of the public health and welfare. . . Such criteria shall . . . reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on health and welfare which maybe expected from the presence of an air pollution agent. . ."

Under the Air Quality Act, the issuance of air quality criteria is a vital step in a program designed to assist the States in taking responsible technological, social, and political action to protect the public from the adverse effects of air pollution.

Briefly, the Act calls for the Secretary of Health, Education, and Welfare to define the broad atmospheric areas of the Nation in which climate, meteorology, and topography, all of which influence the capacity of air to dilute and disperse pollution, are generally homogeneous.

Further, the Act requires the Secretary to define those geographical regions in the country where air pollution is a problem—whether interstate or intrastate. These air quality control regions will be designated on the basis of meteorological, social, and political factors which suggest that a group of communities should be treated as a unit for setting limitations on concentrations of atmospheric pollutants. Concurrently, the Secretary is required to issue air quality criteria for those pollutants he believes may be harmful to health or welfare, and to publish related information on the techniques which can be employed to control the sources of those pollutants.

Once these steps have been taken for any region, and for any pollutant or combination of pollutants, then the State or States responsible for the designated region are on notice to develop ambient air quality standards applicable to the region for the pollutants involved, and to develop plans for action for meeting the standards.

The Department of Health, Education, and Welfare will review, evaluate, and approve these standards and plans, and once they are approved, the States will be expected to take action to control pollution sources in the manner outlined in their plans.

At the direction of the Secretary, the National Air Pollution Control Administration has established appropriate programs to carry out the several Federal responsibilities specified in the legislation.

The Air Quality Act of 1967 requires that ". . . criteria issued prior to enactment of this section November 21, 1967 shall be reevaluated in accordance with the consultation procedure . . . and, if necessary, modified and reissued." *Air Quality Criteria for Sulfur Oxides* was first published in March 1967. This edition reflects the reevaluation, and resulting modification called for by the Act.

In accordance with the Act, a National Air Quality Criteria Advisory Committee was established, having a membership broadly representative of industry, universities, conservation interests, and all levels of government. The committee, whose members are listed following this discussion, provided invaluable advice on policies and procedures under which to issue criteria, and provided major assistance in reevaluating the original document.

With the help of a Subcommittee on Sulfur Oxides, expert consultants were retained to rewrite and edit portions of the document, with other segments being revised by staff members of the National Air Pollution Control Administration. After the initial revisions, there followed a sequence of review by the subcommittee, and by the full committee, as well as by individual reviewers especially selected for their competence and expertise in the many fields of science and technology related to the problems of atmospheric pollution by sulfur oxides. These efforts, without which this document could not have been completed successfully, are acknowledged individually on the following pages.

As also required by the Air Quality Act of 1967, appropriate Federal departments and agencies, also listed on the following pages, were consulted prior to issuing this criteria document. A Federal consultation committee, comprising members designated by the heads of seventeen departments and agencies, reviewed the document, and met with staff personnel of the National Air Pollution Control Administration to discuss their comments.

This Administration is pleased to acknowledge the efforts of each of the persons specifically named, as well as the many not named who contributed to the publication of this volume. In the last analysis, however, the National Air Pollution Control Administration is responsible for its content.

JOHN T. MIDDLETON,
Commissioner, National Air Pollution
Control Administration

NATIONAL AIR QUALITY CRITERIA ADVISORY COMMITTEE

Chairman
DR. JOHN T. MIDDLETON, *Commissioner*
National Air Pollution Control Administration

Dr. Herman R. Amberg
Manager, Manufacturing Services Dept.
Central Research Division
Crown Zellerbach Corp.
Camas, Wash.

Dr. Nyle C. Brady
Director, Agricultural Experiment Station
Cornell University
Ithaca, N.Y.

Dr. Seymour Calvert
Director, Statewide Air Pollution Research Center
University of California, Riverside
Riverside, Calif.

Dr. Adrian Ramond Chamberlain
Vice President
Colorado State University
Fort Collins, Colo.

*Dr. Raymond F. Dasmann
Senior Ecologist
Conservation Foundation
Washington, D.C.

Mr. James R. Garvey
President and Director
Bituminous Coal Research, Inc.
Monroeville, Pa.

Dr. David M. Gates
Director
Missouri Botanical Gardens
St. Louis, Mo.

Dr. Neil V. Hakala
President
Esso Research & Engineering Co.
Linden, N.J.

Dr. Ian T. Higgins
Professor, School of Public Health
The University of Michigan
Ann Arbor, Mich.

Mr. Donald A. Jensen
Executive Engineer
Ford Motor Co.
Dearborn, Mich.

Dr. Herbert E. Klarman
Professor of Public Health Administration and Political Economy
School of Hygiene and Public Health
Johns Hopkins University
Baltimore, Md.

Dr. Leonard T. Kurland
Professor of Epidemiology
Mayo Graduate School of Medicine
Head, Medical Statistics
Epidemiology and Population Genetics
Section, Mayo Clinic
Rochester, Minn.

Dr. Frederick Sargent II
Dean, College of Environmental Sciences
University of Wisconsin
Green Bay, Wis.

Mr. William J. Stanley
Director, Chicago Department of Air Pollution Control
Chicago, Ill.

---

* Resigned, October 14, 1968

CONTRIBUTORS AND REVIEWERS

Dr. Mary O. Amdur
Associate Professor of Toxicology
Department of Physiology
School of Public Health
Harvard University
Boston, Mass.

Dr. Rodney R. Beard
Executive Head, Department
of Preventive Medicine
Stanford University Medical School
Palo Alto, Calif.

Dr. Francis E. Blacet
Emeritus Professor of Chemistry
University of California, Los Angeles
Los Angeles, Calif.

Dr. L. J. Brasser
Head, Atmospheric Pollution Division
Research Institute for
Public Health Engineering
Delft, The Netherlands

Dr. Robert Carrol
Professor of Epidemiology
Albany Medical College
Albany, N.Y.

Dr. Eric J. Cassell
Associate Professor of Community Medicine
The Mount Sinai Hospital
Mt. Sinai School of Medicine
New York, N.Y.

Dr. R. E. Eckardt
Director
Medical Research Division
Esso Research and Engineering Co.
Linden, N.J.

Dr. James G. Edinger
Professor of Meteorology
University of California
Los Angeles, Calif.

Dr. David W. Fassett
Director, Laboratory of Industrial Medicine
Eastman Kodak Co.
Rochester, N.Y.

Dr. Lars Friberg
Chief, Department of Hygiene
Karolinska Institute of Hygiene
Stockholm, Sweden

Dr. John R. Goldsmith
Chief, Environmental Hazards
Evaluation Unit
Department of Public Health
State of California
Berkeley, Calif.

Dr. Leonard Greenburg
Professor of Preventive and
Environmental Medicine
Albert Einstein College of Medicine
New York, N.Y.

Mr. John H. Jacobs
Principal Research Physicist
Bell & Howell Research Center
(Chicago, Ill.)
Pasadena, Calif.

Dr. P. E. Joosting
Medical Service
Research Institute for
Public Health Engineering
Delft, The Netherlands

Dr. Herbert Landesman
Consulting Chemist
Pasadena, Calif.

Mr. Benjanmin Linsky
Professor, Department of Civil Engineering
West Virginia University
Morgantown, W. Va.

Dr. Harold MacFarland
Professor, York University Faculty of
Arts and Sciences
Toronto, Ontario

Dr. Robert Mason
Graduate Department of
Community Planning
University of Cincinnati
Cincinnati, Ohio

Dr. James R. McCarroll
Professor of Preventive Medicine
School of Medicine
University of Washington
Seattle, Wash.

Dr. James N. Pitts, Jr.
Professor of Chemistry
University of California, Riverside
Riverside, Calif.

Mr. Alexander Rihm, Jr., P.E.
Assistant Commissioner
Division of Air Resources
New York State Department of Health
Albany, N.Y.

Dr. Bernard Saltzman
Kettering Laboratory
University of Cincinnati
Cincinnati, Ohio

Mr. Jean J. Schueneman
Chief, Division of Air Quality Control
Maryland State Department of Health
Baltimore, Md.

Dr. William S. Spicer
Director, Division of Respiratory Diseases
University of Maryland School of Medicine
Baltimore, Md.

Dr. Wayne T. Sproull
Consultant in Physics
Pasadena, Calif.

Dr. E. Stephens
Research Chemist
Air Pollution Research Center
University of California, Riverside
Riverside, Calif.

Dr. O. Clifton Taylor
Associate Director
Statewide Air Pollution Research Center
University of California, Riverside
Riverside, Calif.

Dr. Moyer D. Thomas
Editor, Inter-Society Committee Manual of Methods for Ambient Air Sampling and Analysis
Riverside, Calif.

Dr. Paul Urone
Professor
Department of Chemistry
University of Colorado
Boulder, Colo.

Mr. Hans K. Ury
Special Consultant
Environmental Hazards Evaluation Unit
California State Department of Public Health
Berkeley, Calif.

Mr. Ralph C. Wands
Director, Advisory Center on Toxicology
National Research Council
Washington, D.C.

Dr. Richard P. Wayne
Oxford University
London, England
Visiting Professor in Photochemistry
University of California, Riverside
Riverside, Calif.

FEDERAL AGENCY LIAISON REPRESENTATIVES

Department of Agriculture
Kenneth E. Grant
Associate Administrator
Soil Conservation Service

Department of Commerce
Paul T. O'Day
Staff Assistant to the Secretary

Department of Defense
Colonel Alvin F. Meyer, Jr.
Chairman
Environmental Pollution Control Committee

Department of Housing and Urban Development
Charles M. Haar
Assistant Secretary for Metropolitan Development

Department of the Interior
Harry Perry
Mineral Resources Research Advisor

Department of Justice
Walter Kiechel, Jr.
Assistant Chief
General Litigation Section
Land and Natural Resources Division

Department of Labor
Dr. Leonard R. Linsenmayer
Deputy Director
Bureau of Labor Standards

Department of Transportation
William H. Close
Assistant Director for Environmental Research
Office of Noise Abatement

Department of the Treasury
Gerard M. Brannon
Director
Office of Tax Analysis

Federal Power Commission
F. Stewart Brown
Chief
Bureau of Power

General Services Administration
Thomas E. Crocker
Director
Repair and Improvement Division
Public Buildings Service

National Aeronautics and Space Administration
Major General R. H. Curtin, USAF (Ret.)
Director of Facilities

National Science Foundation
Dr. Eugene W. Bierly
Program Director for Meteorology
Division of Environmental Sciences

Post Office Department
Louis B. Feldman
Chief
Transportation Equipment Branch
Bureau of Research and Engineering

Tennessee Valley Authority
Dr. F. E. Gartrell
Assistant Director of Health

Atomic Energy Commission
Dr. Martin B. Biles
Director
Division of Operational Safety

Veterans Administration
Gerald M. Hollander
Director of Architecture and Engineering
Office of Construction

AIR QUALITY CRITERIA FOR SULFUR OXIDES

TABLE OF CONTENTS

Chapter		Page
	Preface	iii
	Introduction	x
1	Physical and Chemical Properties and the Atmospheric Reactions of the Oxides of Sulfur	1
2	Sources and Methods of Measurements of Sulfur Oxides in the Atmosphere	17
3	Atmospheric Concentrations of Sulfur Oxides	31
4	Effects of Sulfur Oxides in the Atmosphere on Materials	49
5	Effects of Sulfur Oxides in the Atmosphere on Vegetation	59
6	Toxicological Effects of Sulfur Oxides on Animals	71
7	Toxicological Effects of Sulfur Oxides on Man	89
8	Combined Effects of Experimental Exposures to Sulfur Oxides and Particulate Matter on Man and Animals	103
9	Epidemiological Appraisal of Sulfur Oxides	113
10	Summary and Conclusions	151
Appendices		
A	Symbols	164
B	Abbreviations	165
C	Conversion Factors	166
D	Glossary	167
	Author Index	172
	Subject Index	175
	Acknowledgements	178

INTRODUCTION

Pursuant to authority delegated to the Commissioner of the National Air Pollution Control Administration, *Air Quality Criteria for Sulfur Oxides* is issued in accordance with Section 107b1 of the Clean Air Act (42 U.S.C. 1857c–2b1).

Air quality criteria are an expression of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects on man and his environment. They are issued to assist the States in developing air quality standards. Air quality criteria are descriptive; that is, they describe the effect that have been observed to occur when the ambient air level of a pollutant has reached or exceeded a specific figure for a specific time period. In developing criteria, many factors have to be considered. The chemical and physical characteristics of the pollutants and the techniques available for measuring these characteristics must be considered, along with exposure time, relative humidity, and other conditions of the environment. The criteria must consider the contribution of all such variables to the effects of air pollution on human health, agriculture, materials, visibility, and climate. Further, the individual characteristics of the receptor must be taken into account. Table A is a listing of the major factors that need to be considered in developing criteria.[1]

Air quality standards are prescriptive. They prescribe pollutant exposures which a political jurisdiction determines should not be exceeded in a specified geographic area, and are used as one of several factors in designing legally enforceable pollutant emission standards.

This document focuses on the sulfur oxides commonly found in the atmosphere—sulfur dioxide, sulfur trioxide, their acids, and the salts of their acids. Other oxides of sulfur are well known in the laboratory, but their presence in the atmosphere has not been demonstrated. Futher, this document considers the effects of the sulfur oxides in conjunction with other pollutant classes, especially particulate matter, where important synergistic effects are observed. (Atmospheric particulate matter is treated in detail in a companion document: *Air Quality Criteria for Particulate Matter.*)

This publication reviews the chemical and physical characteristics of the sulfur oxides, and considers the various analytical methods for measuring them in the atmosphere. Also discussed are the effects of the sulfur oxides on visibility, vegetation, and materials. The toxicological effects of sulfur oxides on animals and on man are considered in separate chapters. Finally, there is a discussion of epidemiological studies that assesses the dose-population response and the response of population subgroups (i.e., children, the elderly, respiratory cripples, etc.) to sulfur oxides and to sulfur dioxide in the presence of particulate matter.

In general, the terminology employed follows usage recommended in the publications style guide of the American Chemical Society. A glossary of terms, list of symbols and abbreviations, list of conversion factors for various units of measurement, author index, and subject index are provided.

The literature has been generally reviewed through June 1968. The results and conclusions of foreign investigations are evaluated for their possible application to the air pollution problem in the United States. This document is not intended as a complete, detailed literature review, and it does not cite every published article relating to sulfur oxides in the ambient atmosphere. However, the literature has been reviewed thoroughly for information related to the development of criteria, and the document not only summarizes the current scientific knowledge of sulfur oxides air pollution, but also points up the major deficiencies in that knowledge and the needs for further research.

---

[1] Calvert S. Statement for air quality criteria hearings held by the Subcommittee on Air and Water Pollution of the U.S. Senate Committee on Public Works, July 30, 1968, published in "Hearings Before the Subcommittee on Air and Water Pollution of the Committee on Public Works, United States Senate (Air Pollution–1968, Part 2)."

The technological and economic aspects of air pollution control are considered in companion volumes to criteria documents. The best methods and techniques for controlling the sources of sulfur oxides emissions, as well as the costs of applying these techniques, are described in: *Control Techniques for Sulfur Oxide Air Pollutants.*

Table A.—FACTORS TO BE CONSIDERED IN DEVELOPING AIR QUALITY CRITERIA

- Properties of Pollution:
  - Concentration
  - Chemical composition
  - Mineralogical structure
  - Adsorbed gases
  - Coexisting pollutants
  - Physical state of pollutant
    - Solid
    - Liquid
    - Gaseous
  - Rate of transfer to receptor domain
- Measurement Methods:
  - Hi-Vol sampler
  - Spot tape sampler
  - Dust fall bucket (rate of deposition)
  - Condensation nuclei counter
  - Impinger (liquid filled)
  - Cascade impactor
  - Electrostatic precipitator
  - Light scattering meter
  - Chemical analysis
  - Gas analysis (non-adsorbing)
  - Adsorbed gas analysis
  - Light scattering or attenuation (Ringelmann or visibility observation)
  - Colored suspension
  - Nucleation of precipitation
  - Stabilization of fog
  - Odor
  - Taste
- Exposure Parameters:
  - Duration
  - Concomitant conditions, such as
    - Temperature
    - Pressure
    - Humidity
- Characteristics of Receptor:
  - Physical characteristics
  - Individual susceptibility
  - State of health
  - Rate and site of transfer to receptor
- Responses:
  - Effects on health (diagnosable effects, latent effects, and effects predisposing the organism to disease)
    - Human health
    - Animal health
    - Plant health
  - Effects on human comfort
  - Soiling
  - Other objectionable surface deposition
  - Corrosion of materials
  - Deterioration of materials
  - Effects on atmospheric properties
  - Effects on radiation and temperature

[illegible] reviewed

are considered

Chapter 1

PHYSICAL AND CHEMICAL PROPERTIES AND THE ATMOSPHERIC REACTIONS OF THE OXIDES OF SULFUR

Table *Page*

Acid Solutions in Equilibrium with Water Vapor at Different Relative Humidities 10

1–3 Scattering Ratios for Various Size Droplets of Sulfuric Acid Mist in Equilibrium with Water Vapor at Various Relative Humidities 11

Chapter 1

PHYSICAL AND CHEMICAL PROPERTIES AND THE ATMOSPHERIC REACTIONS OF THE OXIDES OF SULFUR

A. INTRODUCTION

Sulfur dioxide, sulfur trioxide, and the corresponding acids and salts (sulfites and sulfates) are common atmospheric pollutants which arise mainly from combustion processes. In this chapter, the chemical and physical properties of these substances are discussed in relation to their chemical reactions in the atmosphere and their effect in reducing visibility through the atmosphere, and with respect to the methods employed for their estimation. Analytical methods are described more fully in Chapter 2. More extensive treatments of the chemistry of sulfur oxides are to be found in most works on inorganic chemistry (e.g., references 1 and 2).

B. OCCURRENCE

The oxides SO_2 (sulfur dioxide) and SO_3 (sulfur trioxide), with the corresponding acids H_2SO_3 (sulfurous acid) and H_2SO_4 (sulfuric acid) and the salts of these acids, are well known in atmospheric studies. Other sulfur oxides—SO, S_2O_3, S_2O_7, and SO_4—are known in laboratory studies, but their existence in the atmosphere has not been demonstrated. It has been suggested, however, that S_2O_7 may exist in some atmospheres as a result of the reaction between sulfur dioxide and ozone.[3]

Solid and liquid fossil fuels generally contain appreciable quantities of sulfur, usually in the form of inorganic sulfides and/or sulfur-containing organic compounds. Combustion of the fuel in power plants forms sulfur oxides in the ratio of 40 to 80 parts of sulfur dioxide to 1 part of sulfur trioxide. Aside from naturally occurring oxides of sulfur, the burning of fossil fuels such as coal and petroleum in the United States constitutes the major source of sulfur oxides in the atmosphere.

C. PHYSICAL PROPERTIES OF SULFUR DIOXIDES

Sulfur dioxide is a nonflammable, nonexplosive, colorless gas. In concentrations above 0.3 ppm to 1 ppm in air, most people can detect it by taste; in concentrations greater than 3 ppm it has a pungent, irritating odor to most people.[4-7] The gas is highly soluble in water: 11.3 g/100 ml at 20°C, as compared to 0.004, 0.006, 0.003, and 0.169 g/100 ml for oxygen, nitric oxide, carbon monoxide, and carbon dioxide, respectively. The physical properties of sulfur dioxide are listed in Table 1–1.

Table 1–1.—PHYSICAL PROPERTIES OF SULFUR DIOXIDE

Molecular weight	64.06
Density (gas), g/liter	2.927 at 0°C; 1 atm
Specific (liquid) gravity	1.434 at −10° C
Molecular volume (liquid), ml	44
Melting point, °C	−75.46
Boiling point, °C	−10.02
Critical temperature, °C	157.2
Critcal pressure, atm	77.7
Heat of fusion, Kcal/mole	1.769
Heat of vaporization, Kcal/mole	5.96
Dielectric constant (practical units)	13.8 at 14.5°C
Viscosity, dyne sec/cm^2	0.0039 at 0°C
Molecular boiling point constant, °C/1000g	1.45
Dipole moment, Debye units	1.61

D. CHEMICAL PROPERTIES OF SULFUR DIOXIDES AND SULFUROUS ACID

Sulfur dioxide is a gas under ambient atmospheric conditions and can act as a reducing agent or as an oxidizing agent. Of considerable importance to the problem of air pollution is the ability of the gas to react either photochemically or catalytically with materials in the atmosphere to form sulfur trioxide, sulfuric acid, and salts of sulfuric acid. These atmospheric reactions are discussed separately in Section G.

Laboratory experiments demonstrate that sulfur dioxide may act as an oxidizing or as a reducing agent at room temperature. In the gaseous state, sulfur dioxide oxidizes hyrogen sulfide to form elemental sulfur and water. A catalyst is generally used for this process, which is known as the Claus reaction.

$$2H_2S + SO_2 \longrightarrow 3S + 2H_2O. \qquad (1\text{–}1)$$

As a reducing agent, the gas reacts very slowly with oxygen at 400°C to yield sulfur trioxide, but catalytic oxidation to sulfur trioxide occurs at temperatures as low as room temperature.

$$SO_2 \xrightarrow[\text{catalyst}]{\frac{1}{2}O_2} SO_3. \qquad (1\text{–}2)$$

Catalysts effective in this oxidation include finely divided platinum, charcoal, vanadic oxide (V_2O_5), graphite, chromic oxide, ferric oxide, and the nitrogen oxides. The nitrogen oxides are used as the catalyst in the chamber process of manufacturing sulfuric acid from sulfur dioxide.

Ferrous sulfate ($FeSO_4$) catalyzes the direct oxidation of sulfur dioxides to sulfuric acid in the presence of oxygen and water.

$$H_2O + SO_2 \xrightarrow[FeSO_4]{\frac{1}{2}O_2} H_2SO_4. \qquad (1\text{–}3)$$

Some metal oxides oxidize sulfur dioxide directly to sulfate. Magnesium oxide (MgO), ferric oxide (Fe_2O_3), zinc oxide (ZnO), manganic oxide (Mn_2O_3), cerous oxide (Ce_2O_3), and cupric oxide (CuO) are examples. A sulfide is also formed as a product if the metal ion is not reduced to a lower valence state.

$$\underset{\substack{\text{Magnesium}\\\text{oxide}}}{4MgO} + \underset{\substack{\text{Sulfur}\\\text{dioxide}}}{4SO_2} \longrightarrow \underset{\substack{\text{Magnesium}\\\text{sulfate}}}{3MgSO_4} + \underset{\substack{\text{Magnesium}\\\text{sulfide}}}{MgS} \qquad (1\text{–}4)$$

Lead peroxide (PbO_2) is an active oxidizing agent and is used to obtain an estimate of the amount of oxidizable sulfur compounds in air, e.g.,

$$PbO_2 + SO_2 \longrightarrow PbSO_4. \qquad (1\text{–}5)$$

Hydrogen peroxide (H_2O_2) is used extensively as an oxidizing agent in the analysis of air samples for sulfur dioxide: sulfuric acid is formed, and is estimated conductometrically or by titration.

Sulfur dioxide reacts with the halogens. With chlorine, the product is thionyl chloride ($SOCl_2$). On the other hand, the reaction with iodine in aqueous solution yields sulfuric acid and hydrogen iodide, and the decolorization of a starch-iodine mixture is used in one of the methods for the determination of atmospheric SO_2.

Sulfur dioxide reacts with water to form sulfurous acid (H_2SO_3)

$$SO_2 + H_2O \rightleftharpoons H_2SO_3. \qquad (1\text{–}6)$$

The pure acid is unstable and exists only in aqueous solution. Sulfurous acid can react directly with many organic dyes. The West-Gaeke method for the determination of atmospheric sulfur dioxide takes advantage of this property; pararosaniline is used as the organic dye.

E. PHYSICAL AND CHEMICAL PROPERTIES OF SULFUR TRIOXIDE

Sulfur trioxide in ambient air is either derived from combustion sources directly or from the oxidation of atmospheric sulfur dioxide. Sulfur trioxide may exist in the air as a vapor if the water vapor concentration in the air is low enough; but if sufficient water vapor is present (as there probably always is in ambient air), the sulfur trioxide combines immediately with water to yield sulfuric acid in the form of droplets.

$$SO_3 + H_2O \longrightarrow H_2SO_4. \qquad (1\text{–}7)$$

Sulfuric acid, rather than sulfur trioxide, is thus the compound normally found in the atmosphere. Because of the difficulty of measuring free sulfur trioxide in the air, little is known about how much may be present under various circumstances; presumably, it is present in the unhydrated form only in trace amounts.

Sulfur trioxide is a strong acid and readily converts basic oxides to sulfates. It is also a dehydrating agent. When phosphates, carbonates, perchlorates, and salts of other oxyacids react with sulfur trioxide, the corresponding anhydride of the oxy-acid is formed by extraction of the elements of water. Sulfur trioxide may also act as an oxidizing agent, giving free halogens (except fluorine) with many metal and nonmetal halides. Sulfur trioxide reacts as a Lewis acid with a variety of nitrogen-containing organic ring systems to form addition complexes. Gilbert[8] recently reviewed the numerous and diverse reactions of sulfur trioxide with organic compounds.

F. ATMOSPHERIC REACTIONS OF SULFUR OXIDES

1. Laboratory Investigations

Sulfur dioxide is oxidized in the atmosphere by two main processes: photochemical and catalytic. Gerhard and Johnstone[9] determined that the oxidation of sulfur dioxide in 30 percent sulfuric acid droplets of 0.3-μ diameter in the absence of catalysts proceeds at a rate negligible compared to the rate of photochemical oxidation. They pointed out, however, that even in the absence of catalysts the rate of liquid-phase oxidation in a water fog might be faster than in a photochemical smog if the rate depends upon the total amount of dissolved sulfur dioxide.

Junge and Ryan[10] studied the oxidation of sulfur dioxide in solution and found that essentially no reaction occurred in the absence of a catalyst. When ferric chloride was used as a catalyst, oxidation did take place. The final amount of sulfate formed was only slightly dependent on the concentration of catalyst but was a linear function of sulfur dioxide concentration. Johnstone and Coughanowr[11] estimated from their study of sulfur dioxide oxidation in small droplets that, if manganese sulfate was present as 1-μ crystals, the oxidation rate in fog droplets at 1 ppm SO_2 would be about 1 percent per minute. Both investigators found that manganese salts were more effective catalysts than iron salts. Bracewell and Gall[12] also measured rates of catalytic oxidation of sulfur dioxide in droplets and estimated that, in the presence of ferric or manganous ions, rates of oxidation could be sufficient to account for the sulfuric acid content of urban fogs (assuming sulfur dioxide concentrations of 1750 $\mu g/m^3$ or about 0.6 ppm; see appendix for conversion factors).

The oxidation of sulfur dioxide essentially stopped when the pH of the water droplets approached 2 in Junge and Ryan's experiments,[10] and they suggested that the effect is due, at least in part, to the low solubility of sulfur dioxide in strongly acidic solutions. If ammonia was present in the air to neutralize the acid as it was formed, oxidation of sulfur dioxide continued. Van den Heuvel and Mason[13] found that for given concentrations of ammonia and sulfur dioxide the mass of sulfate formed was proportional to the product of the surface area of the drops and the time of exposure.

The catalytic oxidation of sulfur dioxide may also proceed after adsorption of the gas on the surfaces of suspended solid particles. Smith *et al.*[14] demonstrated preferential chemisorption of sulfur dioxide at ambient concentrations on iron oxide and aluminum oxide aerosols followed by multilayered physical adsorption at higher concentrations. Liberti and associates[15] were unable to desorb sulfur dioxide from atmospheric dust samples and concluded from analyses of these samples that adsorbed sulfur dioxide is either oxidized to sulfate or reacts to form a variety of organic compounds. The interaction of sulfur dioxide with atmospheric aerosols is important also from the point of view of the toxicological effects of such pollutant combinations (see Chapter 8).

Gerhard and Johnstone[9] found the photooxidation rate of sulfur dioxide in air and sunlight to be 0.1 percent to 0.2 percent per hour; the rate did not depend upon the presence of sodium chloride nuclei or nitrogen dioxide, or on changes of relative humidity between 30 percent to 90 percent. Renzetti and Doyle[16] found appreciable formation of H_2SO_4 aerosol by irradiation, at 3130 Å, of sulfur dioxide at concentrations below 1 ppm and at relative humidity below 50 percent. More recently Urone *et al.*[17] investigated reactions of sulfur dioxide in air in the presence of water vapor with irradiation at 3100 Å–4200 Å; their results indicate a faster photooxidation rate when hydrocarbon and nitrogen dioxide are present.

Of primary interest in the photochemical oxidation of sulfur dioxide is the formation of particulate matter in hydrocarbon-nitrogen oxides systems. In the absence of sulfur dioxide, little or no aerosol is formed when mixtures of nitrogen oxides and most hydrocarbons (all at atmospheric concentrations) are irradiated. (Varying amounts of particulate matter are, however, formed when nitrogen oxides are irradiated with any of several particular hydrocarbons such as cyclic olefins.) [18–20] In the absence of nitrogen oxides, Johnstone and Dev Jain [21] obtained particulate matter when they irradiated sulfur oxide with *n*-butane both at 20 mm Hg partial pressure; but Kopczynski and Altshuller [22] were unable to detect any formation of particular matter when sulfur dioxide at atmospheric concentration together with atmospheric concentrations of either olefins or paraffins were irradiated. In fact, Renzetti and Doyle [16] and Dainton and Ivin [23] demonstrated that olefins can suppress the production of particulate matter during irradiation of sulfur dioxide in the absence of nitrogen oxides.

On the other hand, mixtures of olefins, nitrogen dioxide, and sulfur dioxide definitely form an aerosol in the presence of sunlight. Thus a major product of the complex photochemical reaction is sulfuric acid,[16 24–26] which is a hygroscopic material that adsorbs water to form light-scattering droplets of sulfuric acid mist. The density of such a haze obviously will depend on the prevailing relative humidity. Sulfuric acid is sufficiently nonvolatile to self-nucleate even at realistic atmospheric concentrations but this may not be necessary in view of the large numbers of nuclei present even in "clean" air. The exact mechanism by which sulfur dioxide is converted to sulfuric acid mist remains unclear. Even more uncertain is the chemical nature of the aerosol reportedly formed in hydrocarbon-nitrogen oxide systems without sulfur dioxide.

For detailed reviews of photochemical air pollution, the work of Leighton,[27] Altshuller and Buffalini,[28] and Stern [29] should be consulted. The basic spectroscopy and photochemistry of sulfur dioxide are discussed by Calvert and Pitts.[30]

2. Field Investigations

Gartrell *et al.*[31] studied the oxidation of sulfur dioxide in coal-burning power plant plumes. Soluble sulfates were collected on membrane filters, and sulfur dioxide was collected in hydrogen peroxide. The sulfur trioxide concentration in the stack gas was 15 ppm to 40 ppm and the sulfur dioxide concentration was about 2200 ppm. Thus, on a weight basis, the ratio of sulfuric acid to sulfur dioxide as initially about 0.03. In successive samples collected from the plume, the investigators found oxidation rates ranging from zero to 2 percent per minute. Increasing rates of oxidation were observed with increasing relative humidity, and the investigators concluded that moisture within the plume of the ambient strata was the most important factor affecting the rate of oxidation.

Katz [32] made simultaneous collections of two air samples in the Sudbury, Ontario, nickel-smelting area to determine sulfur dioxide and "total sulfur contaminants," which he interpreted as sulfur dioxide, sulfur trioxide, and sulfuric acid. The sample for "total sulfur contaminants" was collected in a dilute solution of sulfuric acid and hydrogen peroxide, and the sulfur dioxide equivalent was determined from electroconductivity measurements. The second sample was collected in a starch-iodine solution so that sulfur dioxide could be determined idometrically. Katz found that (1) the average ratio of sulfur dioxide to total sulfur contaminants or to net gaseous acid was highest when the concentration of gases was highest and (2) the ratio decreased from about 95 percent in 1 hour to 65 percent in 12 hours residence time of the pollutant.[32 33] Although reservations exist about the quantitative nature of these ratios because of a number of apparently uncontrolled or uncontrollable parameters in the experiments, they nevertheless indicate qualitatively that sulfur dioxide is oxidized in the atmosphere. In the experiments, interferences may arise from the possible presence of the acidic gas, nitrogen dioxide, the possible selective removal of basic substances in the air with time, the variable effectiveness of the filter in removing particulate matter and sulfuric acid from the air, etc. Analysis of the data presented by Katz shows that over the relatively narrow range of conditions studied the rate of decrease in the ratio of sulfur dioxide to total sulfur contaminants appears to be independent of concentration of contaminants, of the time of day at which the measurements were made, and of ambient temperature. The rate was about 0.035 percent per minute. From this oxidation rate it follows that, if the initial concentration of sulfur dioxide were 1 ppm (2860 $\mu g/m^3$ at 0°C), the concentration, assuming no dilution, would be approximately 2850 $\mu g/m^3$ after 10 minutes, 2800 $\mu g/m^3$ after 1 hour, and 2300 $\mu g/m^3$ after 10 hours. The corresponding sulfur trioxide (as sulfuric acid) concentrations would be approximately 15 $\mu g/m^3$, 90 $\mu g/m^3$, and 830 $\mu g/m^3$, and the weight ratios of sulfuric acid to sulfur dioxide at the respective times would be approximately 0.005, 0.032, and 0.358.

This rate, obtained from the work of Katz, is much smaller than that reported by Gartrell and associates,[31] perhaps in part because Gartrell had more efficient sulfuric acid collection and perhaps in part because atmospheric conditions were not the same. As noted previously, Gartrell and his associates concluded that moisture within the plume of the ambient air strata is the primary rate-determining factor in the oxidation.

The effect of concentration on reaction rate also must be considered. Whereas Katz' data indicated that the rate of loss of sulfur dioxide was independent of concentration, the sulfur dioxide concentrations of the samples (taken in the open air) generally were less than 2 ppm. Although Gartrell *et al.* did not indicate concentrations of the contaminants in their plume samples, concentrations in such a plume would be much higher than 1 ppm to 2 ppm; at the higher concentrations the reaction rate may be faster and concentration dependent.

3. Particulate Sulfate in Polluted Air

The oxidation of atmospheric sulfur dioxide results in the formation of sulfuric acid and other sulfates that typically account for about 5 percent to 20 percent of the total suspended particulate matter in urban air. In general, as expected, there is a relationship (Commins;[34] see also Chapter 3, Section C) between the concentrations of sulfuric acid or sulfate and sulfur dioxide.

In recent years, some studies have been made of the particle size distribution of suspended atmospheric sulfate. This property determines visibility reduction[35] and is an important factor in physiological responses because of its relation to the degree of penetration and retention of particles in lungs. Roesler *et al.*[36] reported sulfate size distributions in downtown Chicago and Cincinnati based on 24-hour samples collected with cascade impactors. They found values for mass median diameter (MMD), i.e., for equivalent spheres of unit density, that average 0.3μ to 0.4μ in the two cities. These were within the range of average MMD value for total sulfur (0.2μ to 0.9μ) found by Ludwig and Robinson[37] in the Los Angeles and San Francisco Bay areas. From 8-hour samples collected continuously for a week in each of four cities, Wagman *et al.*[38] found values for average sulfate MMD (i.e., 0.42μ in Chicago, Cincinnati, and Fairfax, and 0.60μ in Philadelphia) that were in good agreement with these measurements. They also found that sulfate particle sizes generally increased with increasing relative humidity, whereas sulfate concentration was more closely correlated with absolute humidity. Of particular significance is the fact that all of these investigations showed that a major fraction (generally 80 percent or more) of urban atmospheric sulfate is associated with particles below 2μ in diameter. Suspended sulfate is therefore largely in the respirable fraction of particulate matter and is associated mainly with particles that cause the most pronounced reduction in visibility.

G. EFFECTS OF SULFUR OXIDES ON LIGHT TRANSMISSION IN ATMOSPHERE

One of the most noticeable physical effects of air pollution is the reduced visibility in polluted atmospheres. Comprehensive treatments of the subject include those of Steffens,[39] Middleton,[35] and Robinson.[40] A companion document, *Air Quality Criteria for Particulate Matter,* also discusses the subject in Chapter 3, "Effects of Atmospheric

Particulate Matter on Visibility." Since the meteorological effects, as well as reduction of visibility, are considered in detail in the above document, only the salient features of this problem are summarized here.

1. Reduction of Visibility by Air Pollutants

Visibility in the atmosphere is reduced by the scatter and absorption of visibile radiation by air molecules and aerosol particles. Attenuation by scatter and absorption of the light passing from objects to observer reduces the brightness and contrast between objects with the result that the eye's ability to distinguish objects from their background is reduced.

In addition to reducing visibility by attenuation, aerosols that scatter light efficiently are effective in reducing object contrast and visibility because they also scatter light from the sky and sun into the line of sight of an observer. It is a common observation that dark objects, for example mountain ridges, become progressively lighter in shade as they become more distant. The most distant mountain that one can distinguish is typically almost as light or bright as its sky background. Since particulate sulfur oxides are most effective in scattering light,[27] they represent air pollutants that play an important role in reducing visibility in the atmosphere.

2. Scattering of Light by Sulfuric Acid and Sulfate Particles

The quantitative contribution made by the oxides of sulfur to the total scattering of light in various atmospheres has not been resolved, but sulfuric acid mist and other sulfate particulate matter are recognized as important sources of scattering. The latter arise, as noted earlier, from complex oxidation processes, some of which are photochemical in nature.

Regardless of the mechanism by which the particulate matter is formed, one can, nevertheless, formulate an expression for the distance we can see through the atmosphere. The visual range, L_v, along a given path is defined[41] as the greatest distance a black object may be seen when viewed against the sky at the horizon, and for monodisperse particles and monochromatic light at a threshold contrast of 2 percent, is given by the relation

$$L_v = \frac{3.92}{\sigma} = \frac{3.92}{NAE}, \qquad (1\text{-}8)$$

where σ is the scattering coefficient (per unit path length),
N is the number of particles per unit volume of atmosphere,
A is the cross-sectional area of the particles, and
E is the particles scattering ratio.[40]

The particles scattering ratio, E, is the ratio of the area of the wave front acted on by the particles to the geometric area of the particle. This ratio depends on the particle's refractive index, its shape, and its size relative to the wave-length of the light. Refractive indices of sulfuric acid in equilibrium with water vapor at various relative humidities are given in Table 1–2 and in Table 1–3, values for E when $\lambda = 0.54\mu$ are given for

Table 1–2.—DENSITY, PERCENT SULFURIC ACID, AND REFRACTIVE INDEX OF SULFURIC ACID SOLUTIONS IN EQUILIBRIUM WITH WATER VAPOR AT DIFFERENT RELATIVE HUMIDITIES[42 43]

Relative humidity, percent	H_2SO_4, percent	Density	Refractive index
0	100.00	1.8305	1.440
2	84.41	1.7615	1.434
5	69.44	1.6015	1.421
10	64.45	1.5485	1.414
20	57.76	1.4775	1.407
30	52.45	1.4205	1.399
40	47.71	1.3705	1.393
50	43.10	1.3305	1.387
55	40.75	1.3105	1.384
60	38.35	1.2885	1.381
65	35.80	1.2665	1.378
70	33.09	1.2435	1.374
75	30.14	1.2205	1.370
80	26.79	1.2025	1.366
85	22.88	1.1645	1.362
90	17.91	1.1265	1.356
95	11.08	1.0745	1.347
97.5	7.42	1.0385	1.343
98	4.99	1.0315	1.340

Table 1-3.—SCATTERING RATIOS FOR VARIOUS SIZE DROPLETS OF SULFURIC ACID MIST IN EQUILIBRIUM WITH WATER VAPOR AT VARIOUS RELATIVE HUMIDITIES (INTERPOLATED FROM REFERENCE NO. 44)

	Percent relative humidity										
d[a]	50	55	60	65	70	75	80	85	90	95	98
0.1	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.01
0.2	0.24	0.24	0.23	0.23	0.22	0.22	0.21	0.21	0.20	0.19	0.18
0.3	0.88	0.87	0.85	0.84	0.81	0.80	0.78	0.76	0.73	0.69	0.66
0.4	1.63	1.60	1.58	1.56	1.53	1.50	1.47	1.44	1.40	1.33	1.28
0.5	2.50	2.42	2.43	2.41	2.36	2.32	2.27	2.23	2.17	2.07	1.99
0.6	3.18	3.13	3.11	3.06	3.02	2.93	2.92	2.88	2.81	2.69	2.60
0.7	3.63	3.58	3.52	3.53	3.50	3.46	3.42	3.39	3.34	3.23	3.16
0.8	3.94	3.92	3.91	3.89	3.87	3.81	3.84	3.82	3.78	3.72	3.67
0.9	4.00	4.00	3.99	3.98	3.97	3.98	3.97	3.97	3.95	3.94	3.92
1.0	3.84	3.85	3.85	3.85	3.86	3.87	3.87	3.87	3.88	3.89	3.89
1.1	3.30	3.33	3.31	3.38	3.42	3.45	3.48	3.52	3.58	3.67	3.73
1.2	2.84	2.93	2.98	3.02	3.53	3.14	3.21	3.27	3.35	3.49	3.59
1.3	2.33	2.50	2.54	2.58	2.65	2.71	2.78	2.84	2.93	3.07	3.16
1.4	2.20	2.23	2.22	2.29	2.33	2.38	2.37	2.46	2.52	2.63	2.70
1.5	1.78	1.85	1.81	1.88	1.90	1.92	1.95	1.99	2.05	2.16	2.27
1.6	1.66	1.61	1.62	1.68	1.71	1.72	1.76	1.79	1.86	1.90	2.08
1.7	1.92	1.91	1.91	1.91	1.90	1.90	1.90	1.90	1.90	1.90	1.90
1.8	2.11	2.12	2.10	2.08	2.04	2.01	1.98	1.94	1.89	1.82	1.75
1.9	2.47	2.42	2.39	2.35	2.30	2.25	2.21	2.16	2.08	1.91	1.88
2.0	2.43	2.40	2.43	2.40	2.32	2.33	2.29	2.26	2.20	2.10	2.03

[a] Diameter in microns.

various concentrations of sulfuric acid mist of various particle sizes.

a. Droplet Size

When particles of different sizes and different refractive indices are involved, the equation $L_v = 3.9/NAE$ for visual range, must be modified as follows:

$$L_v = \frac{3.9p}{\Sigma N_{ij} A_{ij} E_{ij}}, \qquad (1\text{–}9)$$

where L_v is the visual range in standard units with a contrast limit (threshold) of 0.02,

i&j identify a class of particles of a given diameter (d) and a given refractive index (n), respectively,

N_{ij} represents the number of ij particles per unit volume,

A_{ij} represents the cross-sectional area of an ij particle,

E_{ij} is the scattering ratio of the ij particle,

p is unity if L_v is in the same units as N and A, e.g., L_v is in meters if N_{ij} is number of particles per cubic meter and A_{ij} is cross-sectional area in square meters, but p is 62.14×10^{-5} if L_v is in miles, N_{ij} is in number of particles per cubic meter, and A_{ij} is in square meters.

Waller and co-workers[45, 46] studied acid droplets in urban air and presented data pertaining to a 39 $\mu g/m^3$ sample of sulfuric acid mist with a mass median diameter of 0.5μ and a geometric standard deviation of 8. The relative humidity at the time the sample was collected was 85 percent. The number of particles of various sizes was calculated from the density of sulfuric acid in equilibrium with water vapor at 85 percent relative humidity, and the mass distribution of the sample.

Given the diameter d_{RH} of particles at a specific relative humidity RH, the diameter $d'_{RH'}$ of particles at relative humidity RH′ can be determined from the relationship

$$d'_{RH'} = d_{RH} \left[\frac{C\rho}{C'\rho'} \right]^{1/3} \qquad (1\text{–}10)$$

where C and C′ are weight percent of H_2SO_4 in droplets at relative humidities RH and RH′, respectively, ρ and ρ' are densities of droplets at relative humidities RH and RH′, respectively.

Equation (1–10) shows that there is a shift of the particle size distribution toward larger sizes with increasing relative humidity. Data from Waller's sample were used to calculate mass median diameters for various relative humidities (Figure 1–1).

b. Number of Droplets Per Cubic Meter

In general, the increase in mass median diameter with increase in relative humidity results in greater numbers of particles (Figure 1–2) in the size range of 0.1μ to 2.0μ, the sizes that cause significant reduction in visual range. The consequences are shown in Figure 1–3 where, for example, it can be seen that at 30 μg/m^3 of sulfuric acid mist the visual range is calculated to be 31 miles at 50 percent relative humidity but only 3.1 miles at 98 percent relative humidity. This result is not inconsistent with the data of Robinson.[40]

c. Use of Sulfur Dioxide/Sulfuric Acid Ratio to Calculate Visual Range Reduction

Since several investigators have reported increasing ratios of sulfuric acid mist to sulfur dioxide with increasing relative humidity, and since correlations between sulfur dioxide and suspended particulate matter also have been shown, it is possible, given the sulfur dioxide concentration and the relative humidity, to calculate visual range by using the following:

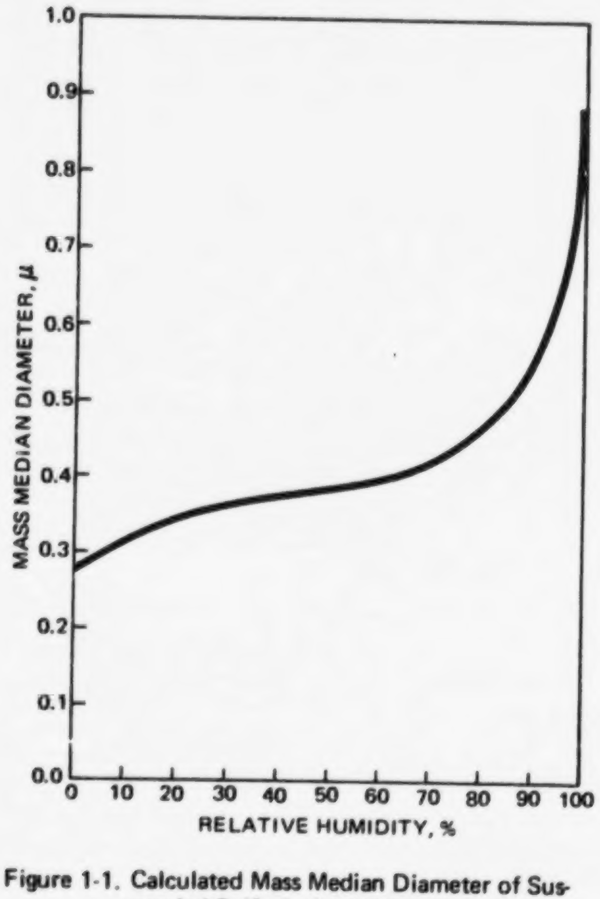

Figure 1-1. Calculated Mass Median Diameter of Suspended Sulfuric Acid Mist Droplets as a Function of Relative Humidity.

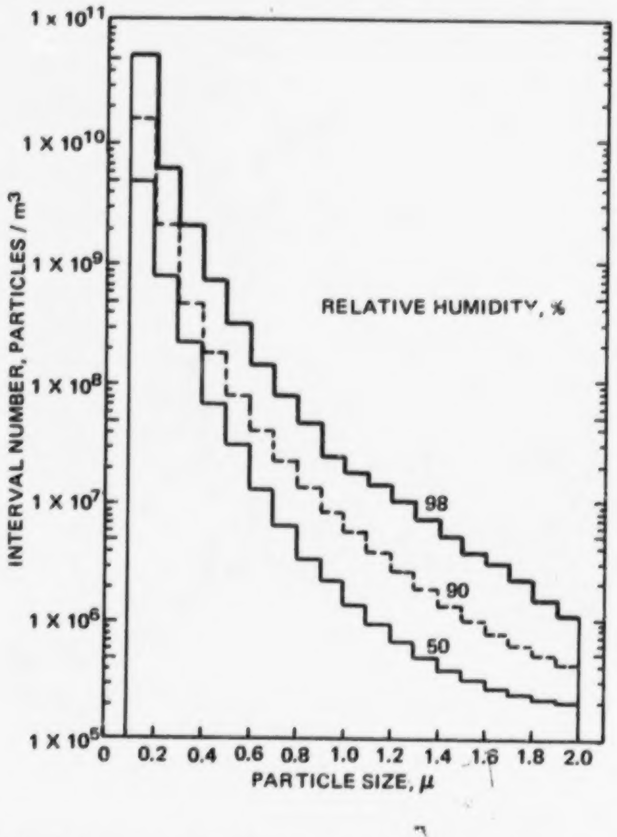

Figure 1-2. Calculated Number of Droplets per m^3 in Various Size Intervals at Different Relative Humidities in a Sulfuric Acid Mist Sample Having a Concentration of 39 μg/m^3.

1. Figure 1–4 shows the average concentrations of sulfuric acid mist (measured as H_2SO_4) associated with various concentrations of sulfur dioxide at various relative humidities as calculated from the ratios reported by Bushtueva.[47 48] Ratios similar to those reported by Bushtueva have also been reported by Coste and Courtier,[49 50] Commins,[51] and Thomas.[52]

2. The average contribution of sulfuric acid mist to the denominator of the visual range equation

$$L_v = \frac{2.4 \times 10^{-3}}{\Sigma N_{ij} A_{ij} E_{ij}} \qquad (1\text{–}11)$$

varies with relative humidities as follows: at 50 percent relative humidity, 0.26×10^{-5} per $\mu g/m^3$; at 90 percent relative humidity, 0.69×10^{-5} per $\mu g/m^3$; and at 98 percent relative humidity, 2.55×10^{-5} per $\mu g/m^3$.

3. From National Air Surveillance Network data for New York City (1964-1965) a typical ratio of suspended particulate matter to sulfur dioxide concentration is 1200 $\mu g/m^3$ to 1 ppm. If the components of the suspended particulate matter other than sulfuric acid are assumed nonhygroscopic, their contribution to the denominator of the visibility equation, determined from the investigations of Charlson,[53] may range from 0.16×10^{-5} to 0.66×10^{-5} per $\mu g/m^3$ with a mean value of 0.33×10^{-5} per $\mu g/m^3$.

For example, in New York City, at 0.3 ppm sulfur dioxide concentration, the suspended particulate matter other than sulfuric acid is 0.3×1200 or $360 \mu g/m^3$. At 90 percent relative humidity the sulfuric acid mist content is $78 \mu g/m^3$.

Then,

$$L_v = \frac{2.4 \times 10^{-3}}{(360)\ (0.33 \times 10^{-5}) + (78)\ (0.69 \times 10^{-5})}$$

$$= \frac{2.4 \times 10^{-3}}{173 \times 10^{-5}} = 1.4 \text{ miles} \qquad (1\text{–}12)$$

The results of a series of such calculations applicable to New York City (1964-1965) are shown in Figure 1–5. Estimates of visual

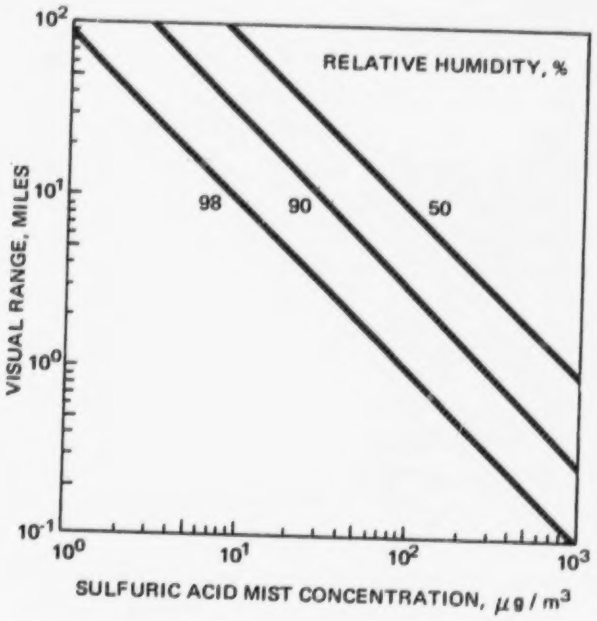

Figure 1-3. Calculated Visibility (Visual Range) in Miles at Various Sulfuric Acid Mist Concentrations and Different Relative Humidities.

This graph shows that visibility decreases with increasing acid mist concentration, and with increasing relative humidity.

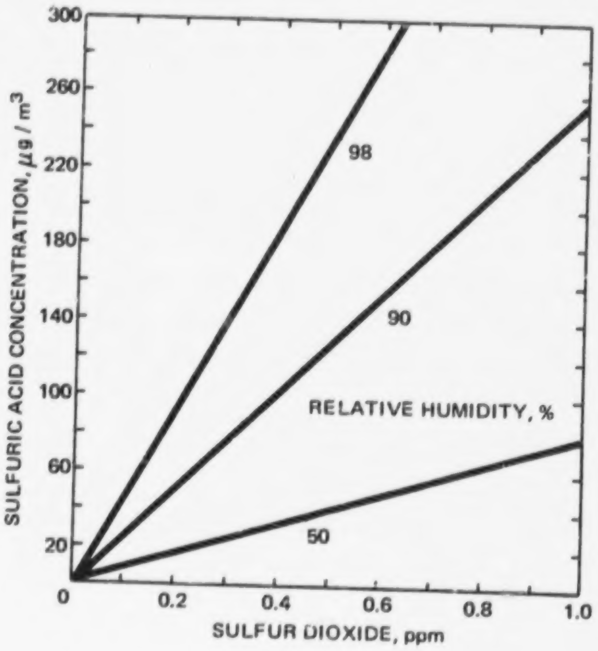

Figure 1-4. Ratios of Sulfuric Acid to Sulfur Dioxide Concentrations at Different Relative Humidities.

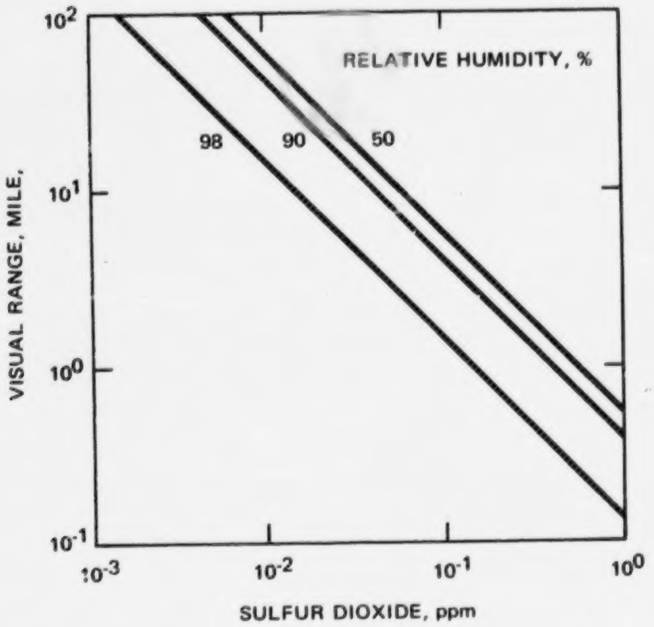

Figure 1-5. Calculated Visibility (Visual Range) in Miles at Various Sulfur Dioxide Concentrations and at Different Relative Humidities in New York City.

This graph shows that visibility decreases with increasing sulfur dioxide concentration, and with increasing relative humidity. It is based on a calculation combining the relationships shown in Figure 1-3 with those shown in Figure 1-4.

range can be obtained for various concentrations of sulfur dioxide. The data become particularly significant in relation to aircraft operations. At a visual range of less than 5 miles, operations are slowed at airports because of the need to maintain larger distances between aircraft; Federal Aviation Administration restrictions on aircraft operations become increasingly severe as the visual range decreases below 5 miles.

H. SUMMARY

The burning of coal and fuel oil, which contain inorganic sulfides and sulfur-containing organic compounds, results in the emission of appreciable quantities of sulfur dioxide into the atmosphere. Other oxides of sulfur are also emitted, but in quantities that are small by comparison; for example, about 40 to 80 parts of sulfur dioxide to one part of sulfur trioxide are emitted from fossil-fueled power plants. Sulfur dioxide is a nonflammable, nonexplosive, colorless gas that most people can taste at concentrations from 0.3 ppm to 1 ppm in air. At concentrations above 3 ppm the gas has a pungent, irritating odor. In the atmosphere, sulfur dioxide, an acid anhydride, is partly converted to sulfur trioxide or to sulfuric acid and its salts by photochemical or catalytic processes. Sulfur trioxide is immediately converted to sulfuric acid in the presence of moisture. Laboratory and field investigations have shown that the oxidation of sulfur dioxide may proceed by several types of mechanism including (1) homogeneous gas phase reactions, (2) homogeneous catalysis in liquids, (3) heterogeneous gas-solid interactions, and (4) heterogeneous gas-liquid interactions. The predominant mechanism and the degree of oxidation are determined by a number of factors, including the concentration, the residence time in the atmosphere, the temperature, the humidity, the intensity and spectral distribution of incident radiation, and the presence of other pollutants such as metal oxides, hydrocarbons, and oxides of nitrogen.

Visibility in the atmosphere is reduced by the scatter and absorption of visible radiation by small particles in the size range from 0.1μ to 1μ in radius. This phenomenon is also described in Chapter 3 of a companion document, *Air Quality Criteria for Particulate Matter*. The attenuation of light from an object and the illumination of the air between the object and the observer reduce the contrast of object and hence reduce its visibility. Of the total suspended particulate matter in urban air, about 5 percent to 20 percent consists of sulfuric acid and other sulfates, and generally 80 percent or more of these particles by weight are smaller than 1 μ radius. Suspended sulfates in the air consequently are very effective in reducing visibility.

The contribution of sulfuric acid mist and other suspended sulfates to the total scattering of light and therefore to reduced visibility can be estimated from data on the concentration and particle size distribution. Generally, a good correlation exists between the concentrations of sulfuric acid or sulfate and the concentrations of sulfur dioxide. Increases in the humidity result in increases in the ratio of sulfuric acid to sulfur dioxide, accompanied by a shift of the mass median diameter of sulfuric acid droplets toward larger sizes and an increase of sulfuric acid concentration in the size range characteristic of acid fogs. Since correlations between sulfur dioxide levels and suspended particulate matter can be found, it is possible for a given relative humidity to estimate the visibility from the sulfur dioxide concentration. The relationship between the visual range, which is the greatest distance a black object may be seen when viewed against the sky at the horizon, and the sulfur dioxide concentration is shown in Figure 1–5 for a ratio of concentrations of sulfur dioxide and particulate matter typical of New York City (1964-1965).

I. REFERENCES

1. Brasted, R. C. "Comprehensive Inorganic Chemistry." Vol. 8, D. Van Nostrand Co., Inc., Princeton, N. J., 1961, 306 pp.
2. Moeller, T. "Inorganic Chemistry." John Wiley and Sons, Inc., New York, 1953, 966 pp.
3. Jacobs, M. B. "Methods for the Differentiation of Sulfur-Bearing Components of Air Contaminants." In: Air Pollution, L. C. McCabe (ed.), McGraw-Hill, New York, 1952, pp. 201–209.
4. Amdur, M. O. "Report on Tentative Ambient Air Standards for Sulfur Dioxide and Sulfuric Acid." Ann. Occup. Hyg., Vol. 3, pp. 71–83.
5. Patty, F. A. (ed.) "Industrial Hygiene and Toxicology. Vol. II. Toxicology." D. W. Fassett and D. D. Irish (eds.), Interscience Pub., Inc., New York, 1963, pp. 831–2377.
6. McCord, C. P. and Witheridge, W. M. "Odors, Physiology and Control." McGraw-Hill, New York, 1949, 53 pp.
7. Bienstock, D., Brunn, L. W., Murphy, E. M., and Benson, H. E. "Sulfur Dioxide—Its Chemistry and Removal from Industrial Waste Gases." U.S. Bureau of Mines, Information Circular 7836, 1958, 22 pp.
8. Gilbert, E. E. "The Reactions of Sulfur Trioxide and of its Adducts, with Organic Compounds." Chem. Rev., Vol. 62, pp. 549–589, 1962.
9. Gerhard, E. R. and Johnstone, G. F. "Photochemical Oxidation of Sulfur Dioxide in Air." Ind. Eng. Chem., Vol. 47, pp. 972–976, May 1955.
10. Junge, C. E. and Ryan, T. "Study of the SO_2 Oxidation in Solution and its Role in Atmospheric Chemistry." Quart. J. Roy. Meteorol. Soc., Vol. 84, pp. 46–55, Jan. 1958.
11. Johnstone, H. F. and Coughanowr, D. R. "Absorption of Sulfur Dioxide from Air. Oxidation in Drops Containing Dissolved Catalysts." Ind. Eng. Chem., Vol. 50, pp. 1169–1172, 1958.
12. Bracewell, J. M. and Gall, D. "The Catalytic Oxidation of Sulfur Dioxide in Solution at Concentrations Occurring in Fog Droplets." Proceedings Symposium on the Physico-chemical Transformation of Sulfur Compounds in the Atmosphere and the Formation of Acid Smogs. Organization for Economic Cooperation and Development, Mainz, Germany, June 1967.
13. Van Den Heuvel, A. P. and Mason, B. J. "The Formation of Ammonium Sulfate in Water Droplets Exposed to Gaseous Sulfur Dioxide and Ammonia." Quart. J. Roy. Meteorol. Soc., Vol. 89, pp. 271–275, April 1963.
14. Smith, B. M., Wagman, J., and Fish, B. R. "Interaction of Airborne Particles with Gases." Presented at Symposium on Colloid and Surface Chemistry in Air and Water Pollution, 156th National Meeting of American Chemical Society, Atlantic City, New Jersey, September 11, 1968. Submitted for publication in Environmental Science and Technology, 1968.
15. Liberti, A. and Devitofrancesco, G. "Evaluation of Sulfur Compounds in Atmospheric Dust." Proceedings of the Symposium on the Physicochemical Transformation of Sulfur Compounds in the Atmosphere and the Formation of Acid Smogs. Organization for Economic Cooperation and Development, Mainz, Germany, June 1967.
16. Renzetti, N. A. and Doyle, G. J. "Photochemical Aerosol Formation in Sulfur Dioxide-Hydrocarbon Systems." Intern. J. Air Pollution, Vol. 2, pp. 327–345, June 1960.
17. Urone, P., Lutsep, H., Noyes, C. M., and Parcher, J. F. "Static Studies of Sulfur Dioxide Reactions in Air." Environ. Sci. Technol., Vol. 2, pp. 611–618, 1968.
18. Shuck, E. A. and Doyle, G. J. "Photooxidation of Hydrocarbons in Mixtures Containing Oxides of Nitrogen and Sulfur Dioxide." Air Pollution Foundation, San Marino, California, Report 29.
19. Doyle, G. J., Endow, N., and Jones, J. L. "Sulfur Dioxide Role in Eye Irritation." Arch. Environ. Health, Vol. 3, pp. 657–667, Dec. 1961.
20. Prager, M. J., Stevens, E. R., and Scott, W. E. "Aerosol Formation from Gaseous Air Pollutants." Ind. Eng. Chem., Vol. 52, pp. 521–524, June 1960.
21. Johnston, H. S. and Dev Jain, K. "Sulfur Dioxide Sensitized Photochemical Oxidation of Hydrocarbons." Science, Vol. 131, pp. 1523–1524, May 20, 1960.
22. Kopczynski, S. L. and Altshuller, A. P. "Photochemical Reactions of Hydrocarbons with Sulfur Dioxide." Intern. J. Air Water Pollution, Vol. 6, pp. 133–135, March–April 1962.
23. Dainton, F. S. and Ivin, K. J. "Photochemical Formation of Sulfinic Acids from Sulfur Dioxide and Hydrocarbons." Trans. Faraday Soc., Vol. 46, pp. 374–381, 1950.

24. Shuck, E. A. Doyle, G. J., and Endow, N. "A Progress Report on the Photochemistry of Polluted Atmospheres." Air Pollution Foundation, San Marino, California, Dec. 1960.
25. Endow, N., Doyle, G. J., and Jones, J. L. "The Nature of Some Model Photochemical Aerosols." J. Air Pollution Control Assoc., Vol. 13, pp. 141–147, April 1963.
26. Goetz, A. and Pueschel, R. F. "Basic Mechanisms of Photochemical Aerosol Formation." Atmos. Environ., Vol. 1, pp. 287–306, 1967.
27. Leighton, P. A. "Photochemistry of Air Pollution." Academic Press, New York, 1961, 300 pp.
28. Altshuller, A. P. and Buffalini, J. J. "Photochemical Aspects of Air Pollution: A Review." Photochem. Photobiol., Vol. 4, pp. 97–146, 1965.
29. Stern, A. C. (ed.) "Air Pollution." 2nd edition, Vols. I and II, Academic Press, New York, 1968.
30. Calvert, J. G. and Pitts, J. N., Jr. "Photochemistry." John Wiley and Sons, Inc., New York, 1966, pp. 209–211.
31. Gartrell, F. E., Thomas, F. W., and Carpenter, S. B. "Atmospheric Oxidation of SO$_2$ in Coal-Burning Power Plant Plumes." Am. Ind. Hyg. Assoc. J., Vol. 24, pp. 113–120, March–April 1963.
32. Katz, M. "Photoelectric Determination of Atmospheric Sulfur Dioxide, Employing Dilute Starch-Iodine Solutions." Anal. Chem., Vol. 22, pp. 1040–1047, 1950.
33. Katz, M. "Sulfur Dioxide in the Atmosphere and its Relation to Plant Life." Ind. Eng. Chem., Vol. 41, pp. 2450–2465, Nov. 1949.
34. Commins, B. T. "Some Studies on the Synthesis of Particulate Acid Sulfate from the Products of Combustion of Fuels and Measurement of the Acid in Polluted Atmospheres." Proceedings of the Symposium on the Physico-chemical Transformation of Sulfur Compounds in the Atmosphere and the Formation of Acid Smogs, Organization for Economic Cooperation and Development, Mainz, Germany, June 1967.
35. Middleton, W. E. K. "Vision through the Atmosphere." Univ. of Toronto Press, Toronto, 1952, 250 pp.
36. Roesler, J. F., Stevenson, H. J. R., and Nader, J. S. "Size Distribution of Sulfate Aerosols in the Ambient Air." J. Air Pollution Control Assoc., Vol. 15, pp. 576–579, 1965.
37. Ludwig, F. L. and Robinson, E. "Size Distribution of Sulfur-Containing Compounds in Urban Aerosols." J. Colloid Sci., Vol. 20, pp. 571–584, 1965.
38. Wagman, J., Lee, R. E., Jr., and Axt, C. J. "Influence of Some Atmospheric Variables on the Concentration and Particle Size Distribution of Sulfate in Urban Air." Atmospheric Environment, Vol. 1, pp. 479–498, 1967.
39. Steffens, C. "Visibility and Air Pollution." In: Air Pollution Handbook, P. L. Magill, F. R. Holden, and C. Ackley (eds.), McGraw-Hill, New York, 1956, pp. 6–1 to 6–43.
40. Robinson, E. "Effects of Air Pollution on Visibility." In: Air Pollution, 2nd edition, Vol. I, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 349–400.
41. Huschke, R. E. (ed.) "Glossary of Meteorology." American Meteorological Society, Boston, Massachusetts, 1959.
42. Lange, N. A. and Forker, G. M. (eds.) "Handbook of Chemistry," 10th edition, McGraw-Hill, New York, 1961, 1969 pp.
43. Weast, R. C., Selby, S. M., and Hodgman, C. D. (eds.) "Handbook of Chemistry and Physics," 45th edition, The Chemical Rubber Co., Cleveland, 1964, 1495 pp.
44. Tendorf, R. B. "New Table of Mie Scattering Functions. Part 6." Geophys. Res. Paper 45, Air Tone Cambridge Research Laboratory, Bedford, Massachusetts, AFCRC-TR-56-20416, 1956.
45. Waller, R. E. "Acid Droplets in Town Air." Int. J. Air Water Pollution, Vol. 7, pp. 773–778, Oct. 1963.
46. Waller, R. E., Brooks, A. G. F., and Cartwright, J. "An Electron Microscope Study of Particles in Town Air." Int. J. Air Water Pollution, Vol. 7, pp. 779–786, Oct. 1963.
47. Bushtueva, K. A. "Ratio of Sulfur Dioxide and Sulfuric Acid Aerosol in Atmospheric Air, in Relation to Meteorological Conditions." Gig. i Sanit., Vol. 11, pp. 11–13, 1954. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey. Vol. 4. Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., Aug. 1960, pp. 193–196.
48. Bushtueva, K. A. "The Determination of the Limit of Allowable Concentration of Sulfuric Acid in Atmospheric Air." In: Limits of Allowable Concentrations of Atmospheric Pollutants. Book 3, 1957. Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., pp. 20–36.
49. Coste, J. H. "Investigation of Atmospheric Pollution." Cong. Int. Quim. Pura Aplicada (Madrid), Vol. 6, pp. 274–287, 1934.
50. Coste, J. H. and Courtier, G. B. "Sulfuric Acid as a Disperse Phase in Town Air." Trans. Faraday Soc., Vol. 32, pp. 1198–1202, 1936.
51. Commins, B. T. "Determination of Particulate Acid in Town Air." Analyst, Vol. 88, pp. 364–367, May 1963.
52. Thomas, M. D. "Sulfur Dioxide, Sulfuric Acid Aerosol and Visibility in Los Angeles." Int. J. Air Water Pollution, Vol. 6, pp. 443–454, Nov.–Dec. 1962.
53. Charlson, R. J., Ahlquist, N. C., and Horvath, H. "On the Generality of Correlation of Atmospheric Aerosol Mass Concentration and Light Scatter." Atmospheric Environment, Vol. 2, pp. 455–464, 1968.

Chapter 2

SOURCES AND METHODS OF MEASUREMENT OF SULFUR OXIDES IN THE ATMOSPHERE

Table of Contents

Page
A. INTRODUCTION 19
B. SOURCES OF ATMOSPHERIC SULFUR OXIDES 19
C. MEASUREMENT OF GASEOUS SULFUR DIOXIDE CONCENTRATIONS 20
  1. Sampling Techniques 20
  2. Colorimetric Method: Pararosaniline 21
  3. Conductometric Methods 21
  4. Acid Titration Method: Hydrogen Peroxide 22
  5. Spectroscopic Methods 22
  6. Other Methods 23
  7. Use and Comparison of Methods 23
D. MEASUREMENT OF SULFURIC ACID AND SULFATES 24
E. OTHER METHODS OF MEASURING POLLUTION BY SULFUR OXIDES 24
  1. Sulfation Rates of Lead Peroxide Candles 24
  2. Suspended Sulfate 25
  3. Sulfate in Dustfall 25
  4. Sulfuric Acid Mist 25
F. SUMMARY 25
G. REFERENCES 27

List of Tables

Table
2-1 Atmospheric Sulfur Dioxide Emissions in 1963 and 1966 by Source 20

Chapter 2

SOURCES AND METHODS OF MEASUREMENT OF SULFUR OXIDES IN THE ATMOSPHERE

A. INTRODUCTION

Oxidized sulfur in the atmosphere exists in several different chemical and physical forms. Under normal conditions, the predominant state is gaseous sulfur dioxide, together with smaller amounts of non-volatile sulfuric acid mist or sulfate salts.

Since the proper and efficient establishment of criteria and standards ultimately depends upon reliable analytical data, it is particularly important that proper attention be given to (1) the sampling techniques, and (2) the analytical procedures employed. These have been reviewed recently by Hendrickson[1] and Katz.[2]

If gaseous sulfur dioxide were the sole pollutant in the atmosphere, its quantitative determination, even in the fractional ppm range, would not be unduly difficult. The presence of both sulfuric acid mist and sulfate salts adds complications to the analytical procedures. These are further complicated by real or potential interferences from a variety of additional air pollutants often found in conjunction with sulfur dioxide.

Some of the methods used for determining SO_2 are *specific*, while others are *general* and depend on a general property such as acidity or conductivity and are subject to errors because of interferences. This has led to some problems in the interpretation of analyses from different laboratories using different techniques.

In the following discussion, the man-made sources of atmospheric oxides of sulfur will be outlined, and the more important methods of their determination, including recent techniques involving remote sensing of SO_2, will be discussed.

Agreement between the various methods of measurement is not always good, because each method is subject to interference from differing causes which may lead to either high or low results.

Sulfuric acid mist is determined by the separation of the mist from sulfur dioxide and the subsequent measurement of the acid or of the sulfate content. Measurements of suspended particulate sulfate and sulfate in dustfall are obtained by means of conventional sulfate determinations.

B. SOURCES OF ATMOSPHERIC SULFUR OXIDES

Sulfur dioxide pollution results primarily from the combustion of fossil fuels, the refining of petroleum, the smelting of ores containing sulfur, the manufacture of sulfuric acid, the burning of refuse, paper making, and the burning or smoldering of coal refuse banks. In all of these processes, a small amount of sulfur trioxide or sulfuric acid also is emitted. Sulfur trioxide is normally present in the atmosphere in extremely small concentrations because it is converted to sulfuric acid soon after its entry into the atmosphere.

Specific reviews covering the coal industry, petroleum refineries, fuel oil combustion, burning coal mine refuse banks, sulfuric acid manufacture, the iron and steel industry, and sulfite pulping have been published.[3–12] Sources of sulfur oxides in the atmosphere also have been reviewed by Rohrman and Ludwig.[13]

Of considerable importance to the meteorological and chemical behavior of sulfur oxides in the atmosphere, as well as to their measurement, are the kinds of emitters, whether large or small and whether disperse or point sources. The trend of operations has been away from sulfur dioxide pollution by low-level disperse sources and toward large point sources, except for space heating and diesel trucks using fuels of high sulfur content. The large source emissions contain lower concentrations of polynuclear hydrocarbons and higher concentrations of nitrogen oxides and sulfuric acid. Particulate matter which interacts with the oxides of sulfur can be controlled to a greater extent when emitted from the larger sources. Further, emissions from large sources usually are emitted from higher stacks and although this practice appears to reduce average ground level concentrations and the frequency of air pollution episodes, [9, 10, 14-17] it may result in early morning fumigations.

Sources of atmospheric sulfur oxides can be considered from the viewpoint of their annual production. Obviously, sulfur-containing coal and fuel oil utilized for heating during the winter will produce a seasonal increase in atmospheric SO_2. This is in contrast with industrial sources, such as power stations and factories, whose effluents are more or less constant throughout the year.

Major sources of sulfur dioxide released to the atmosphere in 1963 [13] and in 1966 [18] are presented in Table 2–1.

C. MEASUREMENT OF GASEOUS SULFUR DIOXIDE CONCENTRATIONS

1. Sampling Techniques

Before discussing specific analytical methods, it is important to recognize that regardless of how accurate the technique may be, the validity of the final results is dependent upon the sampling technique employed for the determination. For example, consideration must be given to factors such as adsorption on, and desorption from, inlet tubes utilized in the sampling apparatus.[19] Teflon *, Tygon *, glass, stainless steel and aluminum have been tested for various lengths and flow rates; in general, a conditioning period is re-

Table 2–1.—ATMOSPHERIC SULFUR DIOXIDE EMISSIONS IN 1963 AND 1966 BY SOURCE

Process	Sulfur dioxide [a]			
	1963		1966	
	Tons	Percent of total emissions	Tons	Percent of total emissions
Burning of coal:				
Power generation (211,189,000 tons, 1963 data)	9,580,000	41.0	11,925,000	41.6
Other combustion (112,630,000 tons, 1963 data)	4,449,000	19.0	4,700,000	16.6
Subtotal	14,029,000	60.0	16,625,000	58.2
Combustion of petroleum products:				
Residual oil	3,703,000	15.9	4,386,000	15.3
Other products	1,114,000	4.8	1,218,000	4.3
Subtotal	4,817,000	20.7	5,604,000	19.6
Refinery operations	1,583,000	6.8	1,583,000	5.5
Smelting of ores	1,735,000	7.4	3,500,000	12.2
Coke processing	462,000	2.0	500,000	1.8
Sulfuric acid manufacture	451,000	1.9	550,000	1.9
Coal refuse banks	183,000	0.8	100,000	0.4
Refuse incineration	100,000	0.4	100,000	0.4
Total Emissions	23,360,000	100.0	28,562,000	100.0

[a] A small amount of this tonnage is converted to sulfuric acid mist before discharge to the atmosphere. The rest is eventually oxidized and/or washed out. Only under unusual meteorologic conditions (Chapter 3) does accumulation occur. The increasing output of sulfur oxides due to increasing power demand is evident.

---

* Mention of commercial products does not constitute endorsement by the National Air Pollution Control Administration.

quired by Tygon and aluminum tubing. For relatively high flow rates (28.3 l/min), aluminu required a conditioning period of about 5 hours at 0.2 ppm and Tygon required a much longer period and should be avoided. Neither adsorption nor desorption was significant in glass or stainless steel tubing at various flow rates for tubing lengths as great as 30.5 meters. Teflon tubing falls into this same class. Any of these three materials can be used for sampling sulfur dioxide at sufficiently high flow velocities (greater than 3.7 m/sec) without prior conditioning and without effect by temperature or humidity.

Both continuous and intermittent sampling are commonly used for sulfur dioxide. To a large extent the type of instrument selected is dependent upon the measurement principle desired. The West-Gaeke and the Conductometric methods are most commonly employed in the United States. Many agencies in the country use intermittent or manual sampling where the collected sample is returned to the laboratory for analysis. Intermittent sampling can be used to provide integrated samples of from 1 hour to 24 hours and is less expensive than continuous monitoring. Continuous monitoring is necessary where it is important to show diurnal changes or the influence of local sources and meteorology.

2. Colorimetric Method: Pararosaniline

In the West-Gaeke method, sulfur dioxide is absorbed in dilute aqueous sodium tetrachloromercurate to form the nonvolatile dichlorosulfitomercurate ion, which then reacts with formaldehyde and bleached pararosaniline to form red-purple pararosaniline methylsulfonic acid. This reaction is specific for sulfur dioxide and sulfite salts. The color intensity of the dye, which is proportional to the concentration of sulfur dioxide, is measured at a wavelength of 560 mμ. The method can be used to determine concentrations in the air from 0.002 ppm to 5 ppm. Ozone and nitrogen dioxide reduce the apparent concentration by destroying some of the dye, although interference by nitrogen dioxide can be eliminated by adding sulfamic acid after sample collection or just prior to analysis. Heavy metal salts, especially iron salts, oxidize dichlorosulfitomercurate, which also results in a lowering of the apparent sulfur dioxide concentration. This effect can be eliminated by a membrane prefilter or by including the disodium ethylenediaminetetraacetic acid in the absorbing reagent to sequester metallic ions. Hydrogen sulfide precipitates mercuric sulfide from the collecting reagent, and such a precipitate must be removed by centrifugation from the sample before proceeding with color development.

Recently two improved West-Gaeke (pararosaniline) methods were developed for the determination of sulfur dioxide in ambient air.[20] These give greater sensitivity and reproducibility, as well as adherence to Beer's Law throughout a greater working range, than does the original West-Gaeke method. The improvements resulted from optimization of several important parameters. Specifically the pararosaniline dye was purified and standardized to reduce variability. Phosphoric acid was used in the final color development to control pH. The pararosaniline methylsulfonic acid produced in the reaction exhibits a hypsochromic spectral shift with increasing pH. Hence the reaction product behaves as a two-color pH indicator, and regardless of the pH condition under which the reaction is carried out, the spectra can be interchanged by addition of acid or base. The sharp peak with an absorbance maximum at $\lambda = 548$ mμ (rose-red) at the higher pH value (1.68) shifts to $\lambda = 575$ mμ (magenta) at a lower pH value (1.02). Interferences from nitrogen oxides, ozone, and heavy metals are minimal, and laboratory results are reproducible to within 4.9 percent (at 95 percent confidence level) if recommended precautions are taken. It is noted that these modifications, although specifically developed for sulfur dioxide in the atmosphere, can be applied to determination of sulfite in other materials. This method has been adopted as the standard or reference method by the NAPCA.

3. Conductometric Methods

The basis of these methods is the oxidation of sulfur dioxide to sulfuric acid by aqueous hydrogen peroxide, and the subsequent measurement of the increased electrical conductivity of the solution. This is a *general* technique and one must take special precautions to eliminate other pollutants which could affect the conductivity of the solution. For example, acidic gases such as the hydrogen halides will increase the conductivity of the solution so that, in the presence of such gases, incorrectly high SO_2 values will be indicated. Weakly acidic gases such as hydrogen sulfide cause practically no intereference because of their slight solubility and poor conductivity, and nitrogen dioxide produces minimum interference because it is poorly absorbed. Sulfuric acid mist is not efficiently collected in the usual gas "scrubber" and therefore is not measured. Salt spray from ice and snow control may give high readings. Ammonia interferes with electroconductivity measurements by neutralizing the acid and forming the ammonium ion which has a high transport number of the opposite charge.

$$\underset{\text{Ammonia}}{2NH_3} + \underset{\text{Sulfuric Acid}}{H_2SO_4} \longrightarrow \underset{\text{Ammonium Sulfate}}{(NH_4)_2SO_4} \qquad (2\text{-}1)$$

Conductivities that are too low are then recorded, since the transport number of all other cations is several times less than that of the hydrogen ion.

Conductometric methods can be automated readily so that one can obtain instantaneous readings. Air is drawn through either acidic hydrogen peroxide solution or through deionized water, and the sulfur dioxide concentration is estimated from the conductivity of the final solution. The interference effects of various gases on the conductivity have been described.[21] [22]

4. Acid Titration Method: Hydrogen Peroxide

In this method, the air sample is bubbled through 0.03N hydrogen peroxide solution adjusted to pH 5. Any sulfur dioxide present forms sulfuric acid, which is then titrated with standard alkali. The presence of other acidic gases in the sample will lead to erroneously high results, and alkaline gases or reactive basic solids give erroneously low results. Generally, a filter is placed in front of the sampling bottle so that no particulate matter or other aerosols are absorbed in the hydrogen peroxide solution. It is important that this be an efficient filter.

This technique is straightforward and the apparatus is inexpensive. It may be automated, if desired. Often, it is used to obtain 24-hour averages, particularly in Europe, where it is widely employed as the standard apparatus and procedure.

5. Spectroscopic Methods

In addition to techniques for making "on-the-spot" analyses for atmospheric sulfur dioxide, it would be a great advantage to have additional methods by which atmospheric concentrations of SO_2 could be determined by means of an instrument remote from the emission source or from the actual air mass being investigated. Recently, there have been developed several spectroscopic techniques which provide such remote sensing capability. Two of the most important are the multiple scan infrared interference spectrometer and the correlation spectrometer. Such instrumentation, though expensive, yields data in a uniquely useful form for a variety of applications.

A commercial model of a multiple-scan infrared interference spectrometer has been developed by Block Engineering Corporation* and its utilization in the field has been discussed by Low.[23] With this instrument, remote detection of SO_2 and CO_2 in the stack effluent of a power plant has been reported by Low and Clancy.[24]

A correlation spectrometer mounted in an airplane recently has been used by Barringer and associates to estimate atmospheric sulfur dioxide "contours" for several U.S. cities.[25-27] The instrumental technique utilizes sunlight reflected from the earth's surface as a "light source" and, in a sophisticated fashion, measures the absorption of this reflected solar radiation by the sulfur dioxide in the air. The method depends on the fact that sulfur dioxide has a clearly structured absorption spectrum in the region near 3100Å and it gives the total sulfur dioxide concentration in the "line of sight" of the spectrometer. While work to date has been carried out primarily from aircraft, correlation spectrometry need not be limited to airborne applications.

---

* Mention of commercial products does not constitute endorsement by the National Air Pollution Control Administration.

6. Other Methods

The earliest methods for determining sulfur dioxide were based on its ability to reduce a starch-iodine solution. This method was considered to be reasonably accurate in the range 0.8 ppm to 3 ppm and it later was modified to cover the range 0.1 ppm to 60 ppm. This method has now been automated.[28] These and other methods are discussed by Katz.[29] He also reported a method of measuring the titratable acidity of the air by absorbing acid components in a water solution of hyperol, a compound formed by reacting hydrogen peroxide with urea. The oxidizable acid components react with the peroxide.

Sulfur dioxide can be determined by a fuchsin-formaldehyde method reviewed by Hochheiser [30] and reported by Alekseeva and Samorodiva.[31] The sample is collected in $0.1N$ sodium hydroxide in glycerol-water solution. Another procedure, reported by Bushtueva,[32] made use of collection in a potassium chlorate solution. The method of analysis was not stated, but it probably was by nephelometric analysis of barium sulfate or turbidimetric analysis of lead sulfate. Lyubimov [33] described a monitoring instrument in which sulfur dioxide is absorbed in a solution of barium chloride, and light transmission through the turbid solution, inversely related to the sulfur dioxide concentration, is recorded.

A further method for determining ozone and sulfur dioxide has been reported recently.[34] The measurement principle involves liberation of iodine by ozone from an iodide solution in one channel of the analyzer and the consumption of iodine by sulfur dioxide in a second analyzer channel.

7. Use and Comparison of Methods

The hydrogen peroxide acid titration method or an automatic conductometric version of the same method is used most frequently in Europe; in the United States, the colorimetric pararosaniline and automated conductometric (using either acidified peroxide or deionized water) methods are used most frequently.

The values obtained by the various methods of measuring gaseous sulfur dioxide in ambient air may not always correlate well with one another, since they do not measure the same thing and because interfering substances are present in varying amounts from time to time and from place to place. It is generally assumed that sulfur dioxide is the major constituent in air that will respond to any of the methods discussed; that the assumption may not be entirely valid is shown in some recent studies.[35–40]

Recently the conductivity and colorimetric pararosaniline techniques have been evaluated in depth,[41, 42] and others have reported on comparisons between the methods.[41, 45] In one study, no consistent relationship between the two measurements was found, although the conductometric method usually gave higher values.[44]

There is evidence that hydrochloric acid may contribute significantly to the values obtained by the hydrogen peroxide method in England, but this may not be true for certain sections of the United States because of the negligible amounts of chloride found in the coal burned.[45–48]

Recently a flame photometric instrument was reported for the detection of total sulfur compounds in the atmosphere.[49] The instrument is based on the principle that when sulfur compounds in air are introduced into a hydrogen-rich flame, they emit light between 300 $m\mu$ to 425 $m\mu$. A photomultiplier tube looks at the light of wavelength 394 $m\mu$ through a narrow-band optical filter. The quantity of light is proportional to the concentration of sulfur compounds. This instrument is readily automated for continuous monitoring.

Several commercial instruments are also available which are based on coulometry. These instruments measure the reducing properties of sulfur dioxide and, with the use of a suitable scrubber, can provide relatively specific and continuous measurements of sulfur dioxide. The principle of this method involves the titration of sulfur dioxide by electrogenerated bromine or iodine.

D. MEASUREMENT OF SULFURIC ACID AND SULFATES

These forms of sulfur are always found in the particulate phase in polluted air, either as suspended matter which can be removed by special measurement techniques or in dustfall. The acidity may be measured by titration or by a related procedure; alternatively, sulfate ion may be measured (frequently by conversion to the insoluble barium salt). Since the particle size is very small, the initial collection of the sample requires special techniques. Any technique for separating particulate matter from air may be used, but filtration and impaction usually have been employed.

A system capable of filtering 50 cubic feet to 60 cubic feet of air in 1 hour through Whatman No. 4 filter paper (1-inch diameter) that had previously been washed with distilled water until the washing had a pH of 7 ± 0.10, is described by Mader.[50] The test filter holding the pollutants was macerated in 20 ml of distilled water, and the pH of the resulting solution was determined. Total acidity (sulfuric acid) was determined by titration of 0.002*N* sodium hydroxide to an end point equivalent to the pH of carbon-dioxide-free distilled water and corrected by a blank determination.

Commins[51] has described a method in which sulfuric acid in the air was collected by filtering air through Whatman No. 1 filter paper. The amount of acid was determined by immersing the collected sample in a known excess of 0.01*N* sodium tetraborate in deionized water of pH 7 and titrating back to pH 7 with 0.01*N* sulfuric acid.

Sulfuric acid aerosol has been collected by impaction or filtration to separate sulfuric acid from sulfur dioxide.[52] The acid is decomposed at controlled temperatures under a nitrogen stream, and the liberated sulfur trioxide is then reduced with hot copper to sulfur dioxide. Sulfur dioxide is determined spectrophotometrically, coulometrically, or by flame photometry. The method measures sulfuric acid in the presence of 10 to 100 times as much sulfur dioxide and inert sulfates. Ammonium sulfate, a primary lung irritant, is also measured as sulfuric acid; however, this sulfate probably originates in the atmosphere from the reaction of sulfuric acid and ammonia. The sensitivity is in the parts per billion range using 1 m^3 of air as a sample. DuBois has described a method in which sulfuric acid is collected on glass fiber filters and titrated following thermal separation at 200° C in a nitrogen atmosphere. This method measures free sulfuric acid and does not include ammonium sulfate or sulfuric acid which reacts with particles on the filter.

E. OTHER METHODS OF MEASURING POLLUTION BY SULFUR OXIDES

1. Sulfation Rates of Lead Peroxide Candles

A method for measuring sulfation involves use of the lead peroxide "candle." A paste made of lead peroxide in a gum tragacanth solution is applied to a cotton gauze wrapped around a glass or porcelain form; the product is the lead peroxide candle. The candle is exposed to the ambient air in a louvered shelter for an extended period of time, usually a month, and the lead sulfate formed in the candle is then determined. It is abvious that the method cannot give any information on short-term variations in sulfur oxides pollution levels and that it is only an empirical estimate of the average concentration. The method measures sulfuric acid, hydrogen sulfide, and other sulfur-containing compounds which can form sulfate. In spite of these limitations, sulfation measurements have correlated well with data on biological effects and the deterioration of materials.

A small, dish-type lead peroxide measuring device termed a "sulfation plate," which can be exposed without a protective shelter, has been described.[53] The plates are more reactive than candles, and it is proposed that exposure periods could be shortened to 1 day with more sensitive analytical methods. Turbidimetric determination of sulfate was found, not unexpectedly, to be less time-consuming than gravimetric analysis, though not as accurate.

The primary application of the lead peroxide candle is for mapping sulfur pollution in a given area as related to sources and meteorology. It is convenient since a simple shelter requiring no electrical power is used. A major problem with the lead peroxide candle is that results are influenced by wind movement and humidity. Another major drawback to the lead peroxide candle is that the laboratory preparation and analysis is quite time consuming. The cost of preparing and analyzing a lead peroxide candle is approximately the same as for collecting and analyzing a 24-hour integrated sulfur dioxide sample by the West-Gaeke procedure. Also, the lead peroxide candle provides intelligence on the oxidizable sulfur compounds in the atmosphere which seldom can be directly related to sulfur dioxide. It has been observed that in many cases monthly averages of sulfur dioxide rise and fall somewhat parallel to monthly sulfation values.

Sulfation rates in United States cities have been observed to range from a few hundredths of a milligram to about 8 mg of sulfur trioxide per 100 cm^2 of exposed lead peroxide candle surface per day (mg SO_3/100 cm^2-day).[35–37, 55]

2. Suspended Sulfate

Sulfate (sulfuric acid and sulfate salts) in suspended particulate matter has been extensively measured for a number of years by the National Air Surveillance Networks.[48, 56] Sulfates are extracted from the particulate matter on the glass fiber filter by refluxing with distilled water. Sulfate in the extract is determined by the methyl thymol blue automated method or by the turbidimetric method. The maximum 24-hour average concentration estimated from 2,197 samples collected over the years 1957 to 1960 is 94 $\mu g/m^3$, and the national average for this period is 11.8 $\mu g/m^3$. There is considerable geographic variation. The observed average concentrations of 18.8 $\mu g/m^3$, 15.0 $\mu g/m^3$, 14.5 $\mu g/m^3$, and 13.3 $\mu g/m^3$ occur in the Mid-Atlantic, Midwest, Mideast, and New England areas. respectively. Lower average concentrations of 10.7 $\mu g/m^3$, 9.0 $\mu g/m^3$, 8.7 $\mu g/m^3$, 7.4 $\mu g/m^3$, and 5.8 $\mu g/m^3$ have been observed in the Southeast, Pacific coast, Great Plains, Gulf south, and Rocky Mountain areas, respectively. As with sulfur dioxide, the highest concentrations occur most frequently in the fall and winter months.

3. Sulfate in Dustfall

The register of Air Pollution Analyses[53] lists only four organizations which have measured and reported sulfate in dustfall in the United States. This measure may be expressed either as percent sulfate in total dustfall or as tons of sulfate per square mile per month. In a southwestern city, where 32 collecting stations were used, sulfate averaged 10.8 percent of the total dustfall, or 15.5 tons/mi^2-month over a 3-month period. In a northwestern city, sulfate averaged 27 percent of the total dustfall, or 7.4 tons/mi^2-month, which is similar to the 32 percent of the total dustfall and 7.8 tons/$mile^2$-month observed in a New England town.[54, 57–59]

4. Sulfuric Acid Mist

Few attempts have been made to measure sulfuric acid mist in the United States. Concentrations measured in Los Angeles average about 25 $\mu g/m^3$, and a high concentration of 50 $\mu g/m^3$ has been observed.[50, 60, 61] In Chicago, during the months of November and December 1964, the average for the hours 10 a.m. to 4 p.m. was 9.2 $\mu g/m^3$.[62]

F. SUMMARY

In 1966, an estimated 28.6 million tons of sulfur dioxide were emitted to the atmosphere, as compared with 23.4 million tons in 1963. The principal share, i.e., 58.2 percent, came from the combustion of coal, most of which was used to generate electrical power. The combustion of residual fuel oil and other petroleum products accounted for 19.6 percent of the total, while the remainder came from the refining of petroleum (5.5 percent), the smelting of sulfur-containing ores (12.2 percent), the manufacturing of sulfuric acid (1.9 percent), the burning of refuse (0.4 percent), and the burning or smoldering of coal refuse banks (0.4 percent). Paper-making and some other industrial operations also contributed minor amounts to the total. In all of these processes, small amounts of sulfur trioxide or sulfuric acid are emitted also.

In the United States, the two most common methods of measurement are the colormetric West-Gaeke (pararosaniline) and the conductometric methods. The former is a specific method and the latter is a general method. The colorimetric West-Gaeke method employs a chemical reaction in which a red-purple dye is produced; the intensity of the dye, which is proportional to the concentration of sulfur dioxide, is then measured. This method is specific for sulfur dioxide and sulfite salts. While nitrogen oxides, ozone, and heavy metal salts in the sample can affect the measurement, there are ways to eliminate these interferences. For intermittent as well as continuous sampling, the modified West-Gaeke (pararosaniline) procedure is the most satisfactory, and it is the standard method of the National Air Pollution Control Administration.

Conductometric methods may employ an acidified peroxide solution in which the electrical conductivity changes directly with the concentration of sulfur dioxide in the absorbing solution. Although conductometric methods can be readily automated to obtain readings over long durations, they are *general* techniques and the user must be fully aware that other pollutants can affect the conductivity of the solution. Since often it is not known what other interfering pollutants may be present in the sample, the results are sometimes very approximate, particularly at high and very low sulfur dioxide concentrations.

The technique most frequently used in Europe is the hydrogen peroxide approach, in which sulfur dioxide forms sulfuric acid which is then titrated with standard alkali; this approach may be considered a *general* method, since the presence of other acidic or alkaline gases in the sample may give erroneously high or low results.

An approach that has been widely used is the lead peroxide candle technique, which determines a "sulfation rate." The method gives integrated values for relatively long periods, but, unlike continuous monitoring instruments, provides no indication of short-term fluctuations. While the lead candle method is inexpensive in terms of equipment, the preparation and analysis costs are about the same as for collecting and analyzing 24-hour integrated sulfur dioxide samples by the West-Gaeke procedure. In general, the disadvantages of lead candles far outweigh their advantages. They are useful only in relatively localized studies where the meteorology and exposure do not vary, and then only if great care is exercised to keep the construction of the candles and the chemistry of the analyses constant. In addition, the lead candle provides only a rough indication of sulfur dioxide concentrations, since it is a general approach that may respond to a large number of oxidizable sulfur-containing compounds found in the atmosphere.

Recently, two long-path spectroscopic techniques have been introduced that sense sulfur dioxide concentrations remotely. A multiple-scan interference spectrometer detects from a distance the characteristic infrared sulfur dioxide emissions in heated plumes; a correlation spectrometer looks at the characteristic absorption by sulfur dioxide that occurs near 3100Å. Although these instruments are complex and expensive, they possess a potential for determining the sulfur dioxide "pollution contours" over large areas of the city, as well as the concentrations at different elevations.

Other techniques are still being perfected and may have much promise. Coulometric instruments involve the titration of sulfur dioxide by electrogenerated bromine or iodine. With the aid of suitable scrubbers, the technique may provide a method for determining sulfur dioxide that is both continuous and relatively specific. A newly developed flame photometric instrument measures total sulfur and is therefore not specific for any one sulfur-containing pollutant. In this method sulfur compounds undergo reduction in a hydrogen-rich flame; the light emitted at a characteristic wavelength is proportional to the total atmospheric sulfur.

Other methods are available for analyzing other sulfur-containing compounds. For example, sulfuric acid aerosol in suspended particulate material may be measured by titration or by controlled decomposition to sulfur dioxide. This then can be measured by a number of methods, including spectrophotometry, coulometry, and flame photometry. Particulate sulfate may be analyzed by spectrophotometric or turbidimetric methods.

Each method is unique in terms of the

measuring time resolution, the costs, the skill and time required for the analysis, and the kinds of pollutants which may cause interference and thus impair the accuracy of the results. The techniques selected, therefore, are usually a compromise. Moreover, a single program often will make use of both general and specific methods. In selecting a technique, it is especially important to consider the degree to which the resulting data can be converted for comparison with results obtained from other instruments and at other time periods.

G. REFERENCES

1. Hendrickson, E. R. "Air Sampling and Quantity Measurement." Air Pollution, 2nd edition, Vol. II, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 3–52.
2. Katz, M. "Analysis of Inorganic Gaseous Pollutants." Air Pollution, 2nd edition, Vol. II, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 53–114.
3. Smith, W. S. "Atmospheric Emission from Fuel Oil Combustion." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS–Pub–999–AP–2, Nov. 1962.
4. "Atmospheric Emissions from Petroleum Refineries." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS–Pub–763, 1960.
5. Schueneman, J. J., High, M. D., and Bye, W. E. "Air Pollution Aspects of the Iron and Steel Industry." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS–Pub–999–AP–1, June 1963.
6. Smith, W. S. and Gruber, C. W. "Atmospheric Emissions from Coal Combustion. An Inventory Guide." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS–Pub–999–AP–24, 1966.
7. "Atmospheric Emissions from Sulfuric Acid Manufacturing Processes." Cooperative Study Project Manufacturing Chemists' Association, Inc., and U.S. Public Health Service, PHS–Pub–999–AP–13, 1965.
8. Stahl, R. W. "Survey of Burning Coal-Mine Refuse Banks." U.S. Bureau of Mines, Information Circular 8209, 1964.
9. Hangebrauck, R. P., von Lehmden, D. J., and Meeker, J. E. "Emissions of Polynuclear Hydrocarbons and Other Pollutants from Heat-Generation and Incineration Processes." J. Air Pollution Control Assoc., Vol. 14, pp. 267–278, July 1964.
10. Cuffe, S. T., Gerstle, R. W., Orning, A. A., and Schwartz, C. H. "Air Pollutant Emissions from Coal-Fired Power Plants, Report 1." J. Air Pollution Control Assoc., Vol. 14, pp. 353–362, Sept. 1964.
11. Hendrickson, E. R. (ed.) "Proceedings Conference on Atmospheric Emissions from Sulfite Pulping." Sanibel Island, April 28, 1966.
12. Cuffe, S. T. and Gerstle, R. W. "Emissions from Coal-Fired Power Plants: A Comprehensive Summary." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS–Pub–999–AP–35, 1967.
13. Rohrman, F. A. and Ludwig, J. H. "Sources of Sulfur Dioxide Pollution." Chem. Eng. Prog., Vol. 61, pp. 59–63, Sept. 1965.
14. Engdahl, R. B. "Combustion in Furnaces, Incinerators and Open Fires." Air Pollution, Vol. II, A. C. Stern (ed.), Academic Press, New York, 1962, pp. 3–39. (See also 2nd edition, Vol. III, in press).
15. Lucas, D. H., Moore, D. J., and Spurr, G. "The Rise of Hot Plumes from Chimneys." Int. J. Air & Water Pollution, Vol. 7, pp. 473–500, Aug. 1963.
16. Lucas, D. H. "The Atmospheric Pollution of Cities." Int. J. Air Pollution, Vol. 1, pp. 71–86, Oct. 1958.
17. Scorer, R. S. and Barret, C. F. "Gaseous Pollution from Chimneys." Int. J. Air Pollution, Vol. 6, pp. 49–63, 1962.
18. Electrical World, *168*(20):101, Nov. 13, 1967.
19. Wohlers, W. C., Trieff, N. M., Newstein, H., and Stevens W. "Sulfur Dioxide Absorption on and Desorption from Teflon, Tygon, Glass, Stainless Steel, and Aluminum Tubings." Atmos. Environ., Vol. 1, pp. 121–130, March 1967.
20. Scaringelli, F. P., Saltzman, B. E., and Frey, S. A. "Spectrophotometric Determination of Atmospheric Sulfur Dioxide." Anal. Chem., Vol. 39, pp. 1709–1719, Dec. 1967.
21. Kuczynski, E. R. "Effects of Gaseous Air Pollutants and the Response of the Thomas SO_2 Autometer." Environ. Sci. Technol., Vol. 1, p. 69, Jan. 1967.
22. Rodes, C. F., Elfers, L. A., Palmer, H. F., and Norris, C. H. Sixty-First Annual Meeting of the Air Pollution Control Association, St. Paul, Minnesota, June 23–27, 1968.
23. Low, M. J. D. "Multiple-Scan Infrared Interference Spectroscopy." J. Chem. Ed., Vol. 43, p. 637, 1966.
24. Low, M. J. D. and Clancy, F. K. "Remote Sensing and Characterization of Stack Gases by Infrared Spectroscopy. An Approach Using Multiple-Scan Interferometry." Environ. Sci. Technol., Vol. 1, pp. 73–74, 1967.
25. Barringer, A. R. and Newberry, B. C. "Remote Sensing Correlation Spectrometry for Pollution Measurements." Ninth Conference on Methods in Air Pollution and Industrial Hygiene, Pasadena, California, Feb. 7–9, 1968.
26. Barringer, A. R. and Newberry, B. C. "Molecular Correlation Spectroscopy for Sensing Gaseous Pollutants." 60th Annual Air Pollution Control Association Meeting, Paper 67/196, 1967.
27. Barringer, A. R. and Shock, J. P. "Progress in the Remote Sensing of Vapors for Air Pollu-

tion, Geologic, and Oceanographic Applications." Proceedings of the 4th Symposium on Remote Sensing of the Environment, April 1966, pp. 779–791.

28. Bokhoven, C. and Niessen, H. G. L. "The Continuous Monitoring of Traces of Sulfur Dioxide in Air on the Basis of Discoloration of the Starch-Iodine Reagent with Prior Elimination of Interfering Compounds." Int. J. Air Pollution, Vol. 10, pp. 223–243, 1966.
29. Katz, M. "Sulfur Dioxide in the Atmosphere of Industrial Areas." In: Effect of Sulfur Dioxide on Vegetation, National Research Council, Ottawa, Canada, 1939, pp. 14–50.
30. Hochheiser, S. "Methods of Measuring and Monitoring Atmospheric Sulfur Dioxide." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS–Pub–999–AP–6, Aug. 1964.
31. Alekseeva, M. V. and Samorodiva, R. Ya. S. "Determination of Sulfur Dioxide Colorimetrically with the Aid of Fuchsin-Formaldehyde Solution." Gig. i Sanit., Vol. 10, p. 42, 1953. In: Limits of Allowable Concentrations of Atmospheric Pollutants, Book 2, Office of Technical Services, Washington, D.C., 1955, pp. 90–95.
32. Bushtueva, K. A. "Ratio of Sulfur Dioxide and Sulfuric Acid Aerosol in Atmospheric Air, in Relation to Meteorological Conditions." Gig. i Sanit., Vol. 11, pp. 11–13, 1954. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey. Vol. 4. Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., Aug. 1960, pp. 193–196.
33. Lyubimov, N. A. "A Nephelometer with an Automatic 24-Hour Device for Continuous Recording of Sulfur Dioxide Concentrations in Atmospheric Air." In: Limits of Allowable Concentrations of Atmospheric Pollutants. Book 5. Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., 1962, pp. 120–127.
34. Schulze, F. "Versatile Combination Ozone and Sulfur Dioxide Analyzer." Anal. Chem., Vol. 38, pp. 298–752, May 1966.
35. McCaldin, R. O. and Bye, W. E. "Air Pollution Appraisal—Seward and New Florence, Pa." U.S. Public Health Service, Robert A. Taft Sanitary Engineering Center, 1961.
36. Stalker, W. W., Dickerson, R. C., and Kramer, G. D. "Atmospheric Sulfur Dioxide and Particulate Matter—A Comparison of Methods of Measurements." Am. Ind. Hyg. Assoc. J., Vol. 29, pp. 68–79, Jan. and Feb. 1963.
37. Farmer, J. R. and Williams, J. D. "Interstate Air Pollution Study, Phase II Project Report, Section III, Air Quality Measurements." Interstate Air Pollution Study St. Louis-East St. Louis Metropolitan Areas. U.S. Dept. of Health, Education, and Welfare, Public Health Service, Dec. 1966.
38. Hochheiser, S., Santner, J. T., and Ludman, W. F. "The Effect of Analytical Method on Indicated Atmospheric SO_2 Concentration." J. Air Pollution Control Assoc., Vol. 16, pp. 266–270, May 1966. (Presented at Annual Meeting, Air Pollution Control Association, Toronto, June 1965.)
39. Greenburg, L. and Jacobs, M. B. "Sulfur Dioxide in New York City Atmosphere." Ind. Eng. Chem., Vol. 48, pp. 1517–1521, Sept. 1956.
40. Tabor, E. C. and Golden, C. C. "Results of Five Years' Operation of the National Gas Sampling Network." J. Air Pollution Control Assoc., Vol. 15, pp. 7–11, Jan. 1965.
41. Terabe, M., Comichi, S., Benson, F. B., Newill, V. A., and Thompson, J. E. "Relationships Between Sulfur Dioxide Concentration Determined by the West-Gaeke and Electroconductivity Methods." J. Air Pollution Control Assoc., Vol. 17, pp. 673–675, Oct. 1967.
42. Shikiya, J. M. and McPhee, R. D. "Multi-Instrument Performance Evaluation of Conductivity Type Sulfur Dioxide Analyzers." Presented at 61st Annual Meeting, Air Pollution Control Association, St. Paul, 1968.
43. Trieff, N. M., Wohlers, H. C., O'Malley, J. A., and Newstein, H. "Appraisal and Modification of West-Gaeke Method for Sulfur Dioxide Determination." J. Air Pollution Control Assoc., Vol. 18, pp. 329–331, May 1968.
44. Booras, S. G. and Zimmer, C. E. "A Comparison of Conductivity and West-Gaeke Analyses for Sulfur Dioxide." J. Air Pollution Control Assoc., Vol. 18, pp. 612–615, Sept. 1968.
45. "Air Quality Data (1963) National Air Sampling Network." U.S. Dept. of Health, Education, and Welfare, Public Health Service, 1965.
46. Gorham, E. "Atmospheric Pollution by Hydrochloric Acid." Quart. J. Roy. Meteorol. Soc., Vol. 84, pp. 274–276, July 1958.
47. Cummins, B. T. "Chemistry in Town Air." Research, Vol. 15, pp. 421–426, Oct. 1962.
48. "Air Pollution Measurements of the National Air Sampling Network, Vol. I. Analyses of Suspended Particulates, 1953–1957." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS–Pub–637, 1958.
49. Stevens, R. K., O'Keeffe, A. E., and Ortman, G. C. Title not known, Environ. Sci. Technol. (In press, 1968.)
50. Mader, P. P., Hamming, W. J., and Bellin, A. "Determination of Small Amounts of Sulfuric Acid in the Atmosphere." Anal. Chem., Vol. 22, pp. 1181–1183, Sept. 1950.
51. Cummins, B. T. "Determination of Particulate Acid in Town Air." Analyst, Vol. 88, pp. 364–367, May 1963.
52. Scaringelli, F. P. and Rehme, K. A. "Determinations of Atmospheric Concentrations of Sulfuric Acid by Spectrophotometry, Coulometry, and Flame Photometry." (To be published in Anal. Chem.)
53. Huey, N. A. "The Lead Peroxide Estimation of Sulfur Dioxide Pollution." J. Air Pollution Control Assoc., Vol. 18, pp. 610–611, Sept. 1968.

54. Kenline, P. A. "In Quest of Clean Air for Berlin, New Hampshire." U.S. Public Health Service, Robert A. Taft Sanitary Engineering Center, Tech. Rept. A62-9, 1962.

55. "Air Pollution Measurements of the National Air Sampling Network, Vol. II. Analyses of Suspended Particulates, 1957-1961." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS-Pub-978, 1962.

56. "Register of Air Pollution Analyses." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS-Pub-610, Vol. I, 1958 and Vol. II, 1961.

57. "Air Pollution in the El Paso, Texas Area. Report on a Two-Year Study under a Community Air Pollution Demonstration Project Grant." El Paso City-County Health Unit, 1959.

58. Tyler, R. G. "Report on an Air Pollution Study for City of Seattle." Univ. of Washington, Environ. Res. Lab., March 15, 1952.

59. Anderson, D. M., Lieben, J., and Sussman, V. H. "Pure Air for Pennsylvania." Pennsylvania Dept. of Health and U.S. Dept. of Health, Education, and Welfare, Public Health Service, Nov. 1961.

60. Chaney, A. L. "Investigations to Detect the Atmospheric Conversion of Sulfur Dioxide to Sulfur Trioxide." Am. Petrol. Inst., Vol. 38, pp. 306-312, 1958.

61. Thomas, M. D. "Sulfur Dioxide, Sulfuric Acid Aerosol, and Visibility in Los Angeles." Int. J. Air Pollution, Vol. 6. pp. 443-454, Nov.-Dec. 1962.

62. Boone, R.E. and Brice, R. M. "Continuous Measurement of Acid Aerosol in the Atmosphere." APCA Paper 65-119. (Presented at Annual Meeting Air Pollution Control Association, Toronto, June 1965.)

BLANK PAGE

Chapter 3

ATMOSPHERIC CONCENTRATIONS OF SULFUR OXIDES

Table of Contents

Page
A. INTRODUCTION 33
B. ATMOSPHERIC CONCENTRATIONS OF SULFUR DIOXIDE 33
  1. Concentrations in Urban Areas 33
  2. Concentrations Related to Point Sources 42
    a. Copper Smelter, Australia, 1962 42
    b. Oil Refinery, 1965 42
    c. Smelter, Canada, 1958 42
    d. Smelter, Trail, Canada, 1931 to 1937 42
    e. Power Plant, 1966 42
    f. Power Plant, Pennsylvannia, 1961 42
C. SULFURIC ACID AND ITS RELATION TO SULFUR DIOXIDE 42
D. ACIDITY OF RAINWATER AND PARTICULATE MATTER 45
E. SUMMARY 45
F. REFERENCES 47

List of Figures

Figure
3–1 Long-time Average Sulfur Dioxide Concentrations (ppm) and Associated Maximum Average Concentrations for Various Time Periods for 12 Sites in 11 Cities 36
3–2 Frequency Distribution of Sulfur Dioxide Levels in Selected American Cities, 1962 to 1967 37
3–3 Annual Average Sulfur Dioxide Concentrations for Eight American Cities 39
3–4 Concentrations of Sulfur Dioxide in Chicago for Various Averaging Times and Frequencies, from December 1, 1963, to December 1, 1964 41

List of Tables

Table
3–1 CAMP Data on Sulfur Dioxide Concentrations (ppm by Volume) 34
3–2 Comparison of Calculated and Observed Maximum Sulfur Dioxide Concentrations for Various Averaging Times in 1964 in Chicago 41
3–3 Concentration of Sulfur Dioxide and Sulfuric Acid Mist Observed in the Air 43
3–4 Concentrations of Sulfur Dioxide and Sulfuric Acid Mist Observed in 1961 in Air over Los Angeles and One of its Suburbs, El Segundo 44

Chapter 3

ATMOSPHERIC CONCENTRATIONS OF SULFUR OXIDES

A. INTRODUCTION

This chapter discusses the short and long-term measured concentrations of sulfur oxides in urban areas and in the vicinity of point sources such as power plants, refineries, and smelters. The relation of sulfur oxides in the atmosphere to the acidity of rainwater and of particulate matter also is reviewed.

B. ATMOSPHERIC CONCENTRATIONS OF SULFUR DIOXIDE

1. Concentrations in Urban Areas

Two major programs sponsored by the U.S. Public Health Service have provided the largest body of data on atmospheric sulfur dioxide concentrations in urban areas. The Continuous Air Monitoring Project (CAMP) use the *electroconductivity* method for SO_2 determination in six large cities; more recently, CAMP has installed and is operating continuous, colorimetric SO_2 monitoring devices at all sites in addition to electroconductivity instruments. The National Air Surveillance Networks (NASN) use the colorimetric (West-Gaeke) method to provide 24-hour-sample data for about 100 locations on a 26-times-per-year basis.

Extensive monitoring programs yield large quantities of data which need to be reduced to a useful form. Maximum or average values alone do not provide a total picture of air quality. Distribution plots, showing the percentage of the time sulfur dioxide concentrations are above certain levels, give a more complete insight into the distribution of sulfur dioxide concentrations for a given site.

The frequency distributions obtained from CAMP measurements (for example, see Zimmer and Larsen[1]) show that sulfur dioxide concentrations are not symmetrically distributed but follow a log-normal distribution. This also has been established by Brasser, Joosting, and van Zuilen.[2] CAMP data are plotted as a frequency ditribution for six cities in Figure 3-2. The lower levels of sulfur dioxide that prevail in Los Angeles and San Francisco, as compared with some Eastern cities, are clearly evident.

Table 3-1 gives sulfur dioxide concentrations, in ppm (determined by the hydrogen peroxide method), for 1962 to 1967 at the CAMP sites in eight cities for six different averaging times: 5 minutes, 1 hour, 8 hours, 1 day, 1 month, and 1 year. For some years, the monitoring systems were not in operation at some locations, and this is indicated by asterisks in the columns. The first column gives the calculated maximum concentration using the approach of Larsen[3] described later in the chapter. Listed in the second and third columns are the geometric mean and the standard geometric deviation, similarly calculated, which describe the statistical distribution of the data. Columns 4 and 5 list the range of maximum values that were recorded during the 6-year period, while the maximum for each year is listed in columns 6 to 11.

From the data shown in Table 3-1, it is apparent that higher maxima are obtained as the averaging time becomes shorter. This is because, for shorter averaging times, the instantaneous fluctuations have much greater influence on the resulting values. This trend is shown quite clearly in Figure 3-1, in which the maximum average concentrations are plotted against the averaging time for different cities. The standard geometric deviation (column 3 of Table 3-1) indicates the degree to which a given set of samples deviate from their geometric mean, and therefore it in-

Table 3-1.—CAMP DATA ON SULFUR DIOXIDE CONCENTRATIONS (ppm by volume)

City and averaging time	1962–1967					Maximum for year						Perct. data avail.
	Calc. annl. max.	Geo mean	SGD	Maxima								
				Max.	Min.	62	63	64	65	66	67	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	
Chicago:												
5 min	3.33	0.104	2.20	1.94	1.11	1.13	1.94	1.62	1.59	1.11	1.62	82
1 hr	1.60	0.111	2.01	1.69	0.86	0.86	1.69	1.12	1.14	0.98	1.11	82
8 hr	0.87	0.118	1.84	1.02	0.45	0.45	0.87	1.02	0.74	0.63	0.80	84
1 day	0.64	0.121	1.75	0.79	0.36	0.36	0.71	0.79	0.55	0.48	0.65	85
1 mo	0.24	0.133	1.44	0.35	0.13	0.18	0.33	0.35	0.27	0.27	0.32	93
1 yr	0.14	0.142	1.00	0.18	0.09	0.10	0.14	0.18	0.13	0.09	0.12	100
Cincinnati:												
5 min	1.85	0.016	2.94	1.15	0.65	0.84	0.99	0.88	1.15	0.70	0.65	82
1 hr	0.70	0.018	2.60	0.57	0.41	0.46	0.48	0.55	0.57	0.41	0.42	82
8 hr	0.31	0.020	2.31	0.38	0.14	0.14	0.23	0.27	0.38	0.18	0.18	83
1 day	0.21	0.021	2.16	0.18	0.10	0.11	0.11	0.14	0.18	0.10	0.13	83
1 mo	0.06	0.025	1.65	0.06	0.04	0.04	0.06	0.06	0.06	0.05	0.04	86
1 yr	0.03	0.029	1.00	0.04	0.02	0.03	0.03	0.04	0.03	0.03	0.02	100
Denver:												
5 min	0.36	0.013	2.11	0.96	0.33	-----	-----	-----	0.95	0.96	0.33	84
1 hr	0.18	0.014	1.94	0.36	0.17	-----	-----	-----	0.36	0.26	0.17	84
8 hr	0.10	0.015	1.79	0.14	0.05	-----	-----	-----	0.14	0.10	0.05	85
1 day	0.07	0.015	1.71	0.06	0.02	-----	-----	-----	0.06	0.05	0.02	86
1 mo	0.03	0.017	1.41	0.03	0.01	-----	-----	-----	0.03	0.02	0.01	92
1 yr	0.02	0.018	1.00	0.02	0.01	-----	-----	-----	0.02	0.01	-----	100
Los Angeles:												
5 min	0.47	0.013	2.28	0.68	0.24	0.24	0.51	0.68	-----	-----	-----	59
1 hr	0.22	0.014	2.07	0.29	0.13	0.13	0.19	0.29	-----	-----	-----	67
8 hr	0.12	0.015	1.90	0.13	0.08	0.08	0.09	0.13	-----	-----	-----	71
1 day	0.09	0.015	1.80	0.10	0.06	0.06	0.07	0.10	-----	-----	-----	71
1 mo	0.03	0.017	1.46	0.03	0.02	0.03	0.03	0.02	-----	-----	-----	64
1 yr	0.02	0.018	1.00	0.02	0.02	0.02	0.02	-----	-----	-----	-----	67
Philadelphia:												
5 min	2.60	0.055	2.40	1.25	0.79	1.25	1.05	1.00	1.11	0.79	1.12	75
1 hr	1.16	0.060	2.17	1.03	0.66	1.03	0.85	0.84	0.94	0.66	0.77	76
8 hr	0.60	0.064	1.98	0.71	0.43	0.56	0.58	0.54	0.71	0.43	0.56	77
1 day	0.42	0.067	1.87	0.46	0.35	0.35	0.46	0.43	0.35	0.36	0.35	79
1 mo	0.15	0.075	1.50	0.15	0.12	0.13	0.12	0.15	0.13	0.13	0.13	86
1 yr	0.08	0.081	1.00	0.10	0.06	0.09	0.06	0.09	0.08	0.09	0.10	100
St. Louis:												
5 min	2.40	0.028	2.75	1.42	0.85	-----	-----	1.16	1.42	1.25	0.85	81
1 hr	0.96	0.031	2.45	0.96	0.55	-----	-----	0.73	0.96	0.84	0.55	81
8 hr	0.45	0.034	2.20	0.36	0.24	-----	-----	0.31	0.36	0.33	0.24	82
1 day	0.30	0.036	2.06	0.26	0.18	-----	-----	0.26	0.19	0.18	0.21	84
1 mo	0.09	0.042	1.60	0.08	0.05	-----	-----	0.08	0.06	0.06	0.05	88
1 yr	0.05	0.047	1.00	0.06	0.03	-----	-----	0.06	0.05	0.04	0.03	100
San Francisco:												
5 min	0.56	0.005	2.87	0.33	0.16	0.16	0.33	0.26	-----	-----	-----	64
1 hr	0.22	0.006	2.54	0.26	0.11	0.11	0.26	0.16	-----	-----	-----	64
8 hr	0.10	0.007	2.27	0.10	0.06	0.06	0.07	0.10	-----	-----	-----	65
1 day	0.07	0.007	2.12	0.08	0.05	0.05	0.05	0.08	-----	-----	-----	65
1 mo	0.02	0.008	1.63	0.03	0.01	0.01	0.02	0.03	-----	-----	-----	75
1 yr	0.01	0.010	1.00	0.02	0.01	-----	0.01	0.02	-----	-----	-----	67
Washington:												
5 min	1.19	0.039	2.17	0.87	0.42	0.42	0.56	0.87	0.44	0.47	0.44	69
1 hr	0.58	0.042	1.99	0.62	0.35	0.38	0.48	0.62	0.35	0.45	0.37	69
8 hr	0.32	0.044	1.83	0.35	0.22	0.23	0.35	0.32	0.27	0.34	0.22	70
1 day	0.23	0.046	1.74	0.25	0.15	0.18	0.25	0.22	0.20	0.25	0.15	71
1 mo	0.09	0.050	1.43	0.11	0.07	0.10	0.11	0.09	0.08	0.10	0.07	75
1 yr	0.05	0.050	1.00	0.05	0.04	0.05	0.05	0.04	0.05	0.04	-----	83

Table 3–1 (continued).—CAMP DATA ON SULFUR DIOXIDE CONCENTRATIONS (ppm by volume)

City and averaging time	Percent of time concentration is exceeded								
	.001	0.01	0.1	1	10	30	50	70	90
	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)
Chicago:									
5 min	1.75	1.34	1.03	0.68	0.33	0.16	0.08	0.03	0.01
1 hr	-------	1.12	0.96	0.65	0.33	0.16	0.08	0.03	0.01
8 hr	-------	-------	0.76	0.57	0.32	0.16	0.09	0.04	0.01
1 day	-------	-------	-------	0.50	0.31	0.16	0.09	0.05	0.02
1 mo	-------	-------	-------	-------	0.27	0.17	0.09	0.06	0.03
1 yr	-------	-------	-------	-------	-------	-------	0.12	-------	-------
Cincinnati:									
5 min	0.90	0.70	0.42	0.19	0.07	0.03	0.02	0.01	0.00
1 hr	-------	0.53	0.35	0.17	0.07	0.03	0.02	0.01	0.00
8 hr	-------	-------	0.22	0.12	0.06	0.04	0.02	0.02	0.01
1 day	-------	-------	-------	0.10	0.06	0.04	0.03	0.02	0.01
1 mo	-------	-------	-------	-------	0.05	0.04	0.03	0.02	0.01
1 yr	-------	-------	-------	-------	-------	-------	0.03	-------	-------
Denver:									
5 min	0.64	0.33	0.18	0.07	0.03	0.01	0.01	0.00	0.00
1 hr	-------	0.26	0.11	0.06	0.03	0.02	0.01	0.00	0.00
8 hr	-------	-------	0.08	0.04	0.03	0.02	0.01	0.00	0.00
1 day	-------	-------	-------	0.04	0.03	0.02	0.01	0.00	0.00
1 mo	-------	-------	-------	-------	0.02	0.02	0.01	0.01	0.00
1 yr	-------	-------	-------	-------	-------	-------	0.01	-------	-------
Los Angeles:									
5 min	0.56	0.38	0.15	0.08	0.04	0.02	0.01	0.01	0.00
1 hr	-------	0.25	0.13	0.08	0.04	0.02	0.01	0.01	0.00
8 hr	-------	-------	0.10	0.07	0.04	0.02	0.02	0.01	0.00
1 day	-------	-------	-------	0.06	0.04	0.02	0.02	0.01	0.00
1 mo	-------	-------	-------	-------	0.02	0.02	0.02	0.01	0.01
1 yr	-------	-------	-------	-------	-------	-------	0.02	-------	-------
Philadelphia:									
5 min	1.18	0.95	0.72	0.47	0.21	0.09	0.05	0.03	0.01
1 hr	-------	0.85	0.66	0.45	0.21	0.09	0.05	0.03	0.01
8 hr	-------	-------	0.49	0.37	0.19	0.10	0.06	0.04	0.01
1 day	-------	-------	-------	0.29	0.18	0.10	0.07	0.04	0.02
1 mo	-------	-------	-------	-------	0.12	0.10	0.08	0.07	0.03
1 yr	-------	-------	-------	-------	-------	-------	0.09	-------	-------
St. Louis:									
5 min	1.23	0.96	0.58	0.29	0.11	0.05	0.02	0.01	0.00
1 hr	-------	0.73	0.50	0.25	0.11	0.05	0.03	0.01	0.00
8 hr	-------	-------	0.29	0.19	0.09	0.06	0.04	0.02	0.00
1 day	-------	-------	-------	0.14	0.09	0.05	0.04	0.02	0.01
1 mo	-------	-------	-------	-------	0.06	0.05	0.04	0.03	0.02
1 yr	-------	-------	-------	-------	-------	-------	0.04	-------	-------
San Francisco:									
5 min	0.30	0.18	0.13	0.07	0.03	0.01	0.01	0.00	0.00
1 hr	-------	0.16	0.11	0.07	0.03	0.01	0.01	0.00	0.00
8 hr	-------	-------	0.09	0.06	0.03	0.02	0.01	0.00	0.00
1 day	-------	-------	-------	0.05	0.03	0.02	0.01	0.00	0.00
1 mo	-------	-------	-------	-------	0.02	0.01	0.01	0.01	0.00
1 yr	-------	-------	-------	-------	-------	-------	0.01	-------	-------
Washington:									
5 min	0.65	0.49	0.36	0.22	0.11	0.06	0.03	0.02	0.01
1 hr	-------	0.46	0.35	0.21	0.10	0.06	0.03	0.02	0.01
8 hr	-------	-------	0.26	0.19	0.10	0.06	0.04	0.02	0.01
1 day	-------	-------	-------	0.17	0.10	0.06	0.04	0.02	0.01
1 mo	-------	-------	-------	-------	0.09	0.05	0.04	0.03	0.02
1 yr	-------	-------	-------	-------	-------	-------	0.04	-------	-------

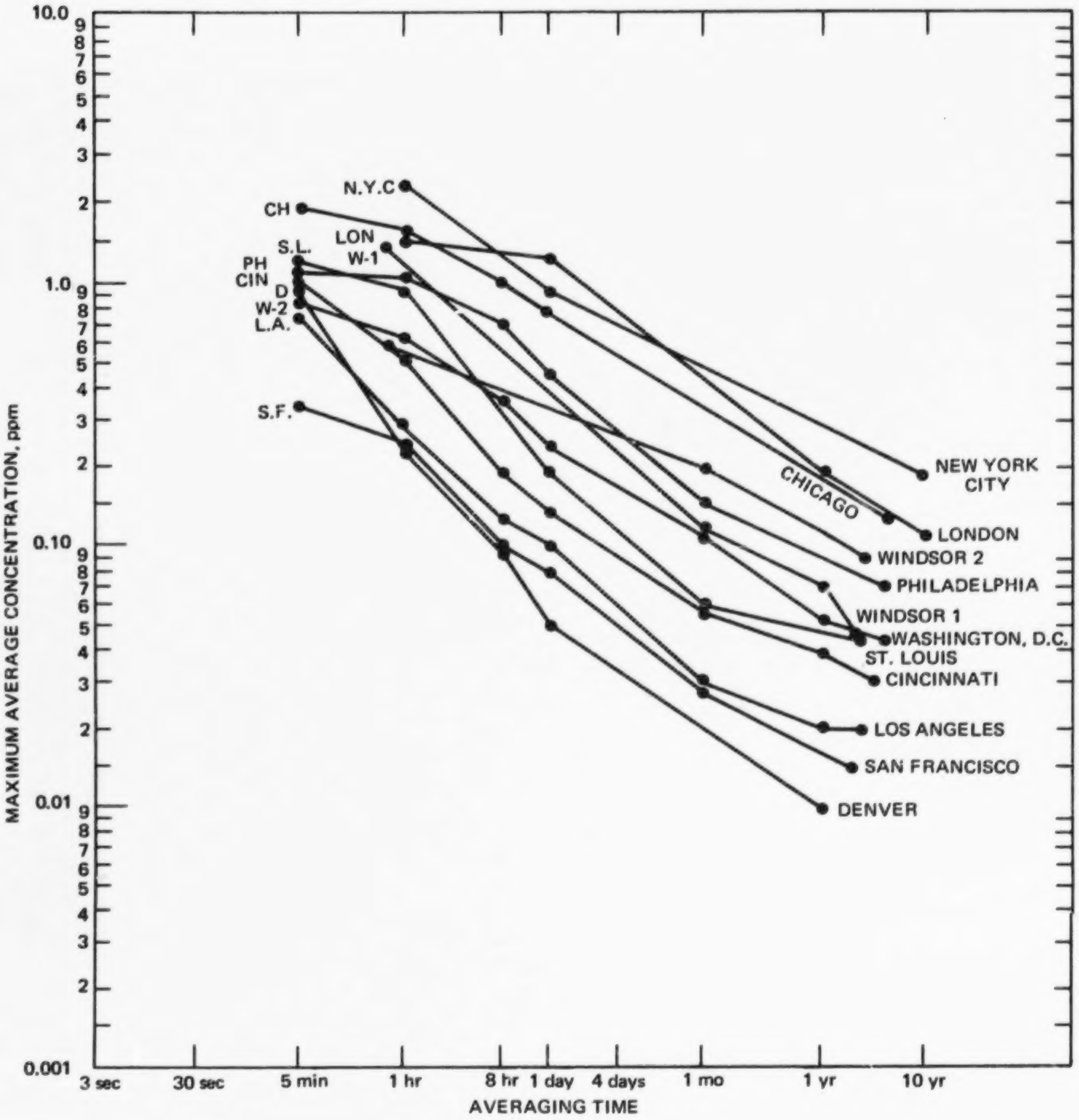

Figure 3-1. Long-time Average Sulfur Dioxide Concentrations (ppm) and Associated Maximum Average Concentrations for Various Time Periods for Twelve Sites in Eleven Cities.

For each of the given lines, the last point on the right is the long-term average over the interval specified, while the other points represent the maxima within the interval for the averaging periods shown. If, for example, one complete year of data were available in terms of hourly averages, there would be one maximum hourly value for that year. If five years of data were available, in terms of hourly averages, five maxima (one for each year) would be obtained, and the highest of these five maxima would constitute the maximum hourly average for the five-year period.

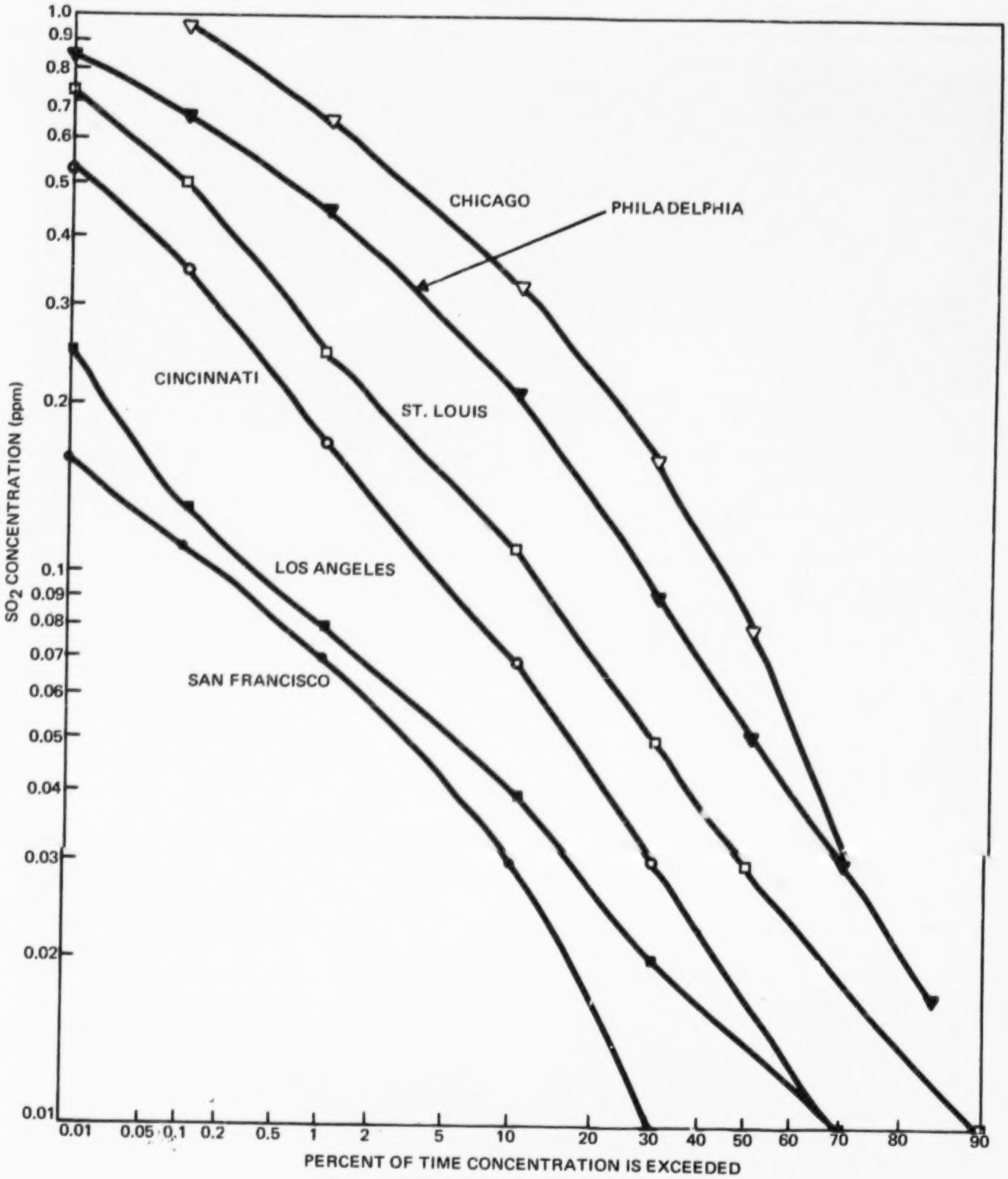

Figure 3-2. Frequency Distribution of Sulfur Dioxide Levels in Selected American Cities, 1962 to 1967 (1-Hour Averaging Time, Data From Table 3-1).

The approximate log normality of the distribution of sulfur dioxide concentrations is shown by the rather straight lines of the distribution functions when these are plotted on a logarithmic scale (concentrations) against a normal distribution frequency.

creases as the time period becomes shorter. For 1-year averages, the geometric mean coincides with the arithmetic mean (standard geometric deviation of unity). For 1-hour-duration samples, the standard geometric deviation varies from about 2 (1.94 in Denver) to 2.6 (in Cincinnati). When the standard geometric deviation for 1-hour-duration samples is 2, the expected annual maximum for 1-hour-duration samples is about 10 times the annual mean. When the standard geometric deviation is 2.5, as it is for San Francisco, the 1-hour maximum is about 20 times the annual mean. Thus, for the data shown, the highest 1-hour average concentration is expected to be from about 10 to 20 times the annual mean as the deviation varies from 2 to 2.5. Therefore, if a 1-hour average concentration is not to exceed 0.3 ppm more than once a year, the annual mean associated with this concentration would be between 0.015 ppm and 0.03 ppm, depending on whether the standard geometric deviation for 1-hour averages is 2.5 or 2, respectively. Similarly, the maximum 24-hour concentration encountered once a year is expected to be between about 4 and 7 times the annual mean concentration for the cities shown, and the monthly maximum ranges from 1.5 to 2 times the annual average. Other cities might have deviations lying outside these boundaries, and, for such cases, the maxima for different time periods and the other relevant parameters could be calculated.[3]

Data from the NASN stations, which cover more cities than the CAMP network, are based on measurements over 24 hours made on 20 to 26 selected dates throughout the year. The highest NASN annual average (0.17 ppm) was recorded in New York City, which also experienced the highest 24-hour average (0.38 ppm); the lowest NASN annual average (0.002 ppm) was in Kansas City, Mo., while the lowest 24-hour averages were below the minimum value detectable by the instruments, i.e., below about 0.001 ppm. In general, at a typical NASN station, the maximum 24-hour average is about 3 times as high as the annual average, but at some stations can be as high as 6 to 8 times the annual averages. These ranges are similar to those of the CAMP stations in Table 3-1. Geographically, the highest values are recorded at stations in cities in the northeastern part of the United States, especially those east of the Mississippi River and north of the Ohio River. In these areas, the major source of energy is fossil fuels of high sulfur content, such as domestically-mined coal and imported residual oils. Maxima in the diurnal concentration curve usually occur about 8 a.m.

Figure 3-1 is a graphical counterpart of Table 3-1, without the frequency distributions but with four stations added (New York City, London,[5] and two stations in Windsor, Ontario).[6] For each of the 12 stations, maximum average concentrations are shown for various averaging times. Since the averaging times are not comparable in some cases, the time points for which maxima are available have been connected in order to approximate the relationship between the corresponding maxima. As expected, a station with a relatively high maximum concentration for one averaging time is likely to show, in proportion, an equally high maximum concentration for other averaging times. A station with a low maximum concentration for one averaging time likewise shows an equally low maximum concentration for other averaging times.

The last nine columns of Table 3-1 give the frequency distribution of concentrations for each of these averaging times (or subsets of these values; for example, for 1 year, with at most six measurements available, only the median, or 50-percentile concentration, is given). While the maximum of columns 6 to 11 were tabulated on a year-by-year basis, the frequency data shown here (columns 12 through 20) are applicable to the entire 6-year period. The value listed in a given column is the concentration which is exceeded the stated percentage of the time. For example, the values listed in the 0.1 percentile columns are those concentrations which were exceeded 0.1 percent of the time; this means that, for 99.9 percent of the time, the concentrations measured were less than or equal to this value. In general, this value is different for different averaging times. At the lower percentiles, this value increases as the averaging time is shortened; in Chicago, for example, the 0.1 percentile concentration changes from 0.76 ppm to 1.03 ppm as the averaging interval is shortened from 8 hours to 5 minutes. At the higher percentiles, the opposite occurs: the concentration decreases as the averaging time is shortened. The 30-percentile point (which is very close to the arithmetic mean) apparently represents an equilibrium condition, since the concentration associated with this point is approximately the same for all averaging times. Thus, the 30-percentile concentration, i.e., the value which is not exceeded for 70 percent of the time, might be used as a simple indicator of the "general sulfur dioxide level" of a city, irrespective of the averaging time. For the eight cities tabulated, using this index, Chicago clearly shows the highest level (0.16 ppm), followed by Philadelphia, Washington, St. Louis, Cincinnati, Los Angeles, Denver, and San Francisco. That this is a good index is borne out by Figure 3-3, which shows the annual average sulfur dioxide concentrations, by year, for these same cities. The ordering of the cities, from most severe to least severe, appears to be the same, with Chicago on the top and San Francisco on the bottom.

The conversion of the maximum concentration for one averaging time to that for another may be approached in a number of

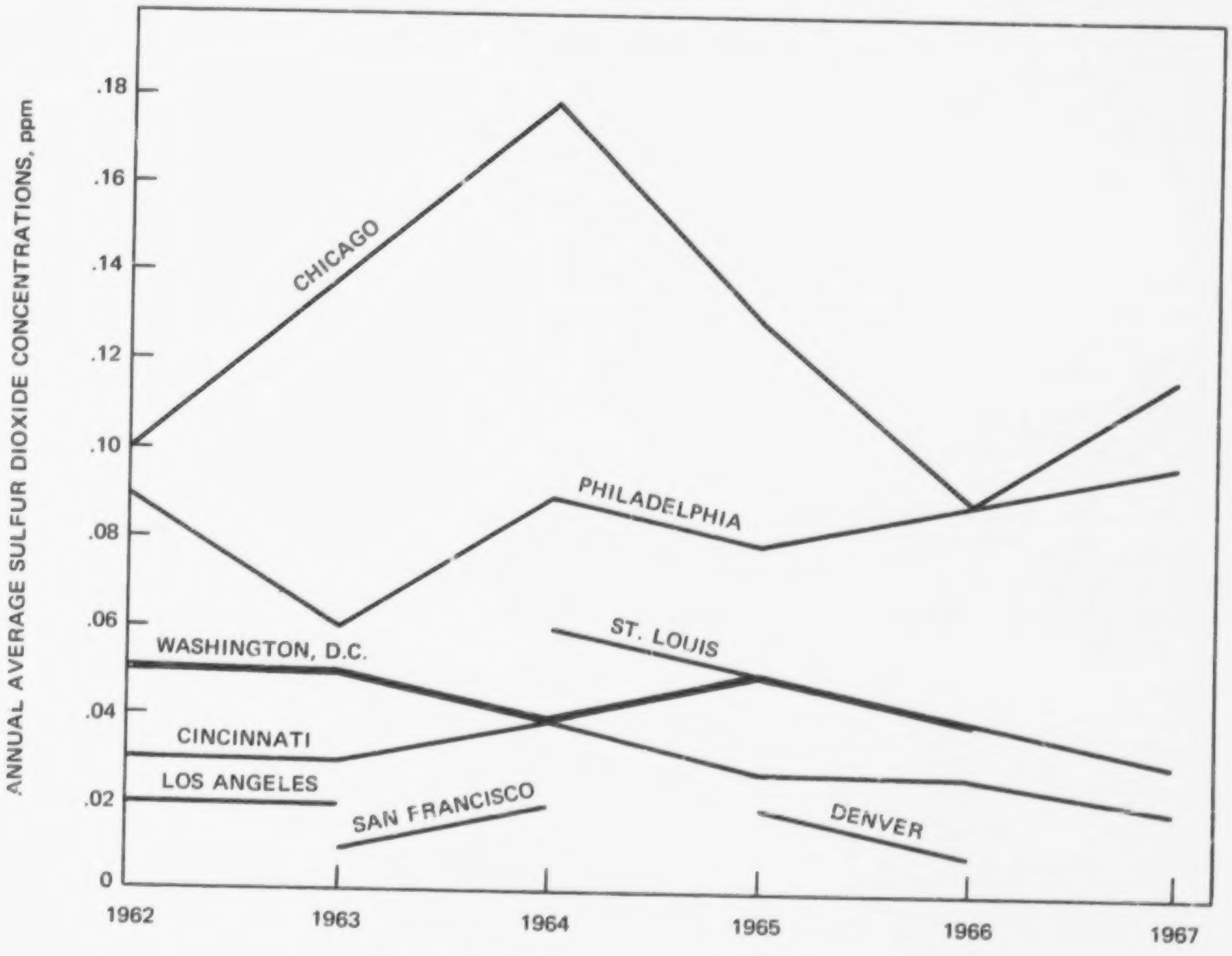

Figure 3-3. Trends in Sulfur Dioxide Concentration for Eight Cities.

This figure shows that the sulfur dioxide concentrations for the years 1962-1967 were highest in Chicago and Philadelphia. (Sampling periods begin on December 1 of the preceeding year and run until November 30 of the year shown).

ways. Brasser, Joosting, and van Zuilen[2], in a manner similar to that described above, attempt to establish empirically the relationship between maxima for different averaging times from measurements carried out over long periods and under different meteorological conditions.

Larsen[3] computes the expected maximum concentration (C) for a given averaging time by using the geometric mean (M_g), the standard geometric deviation (σ_g), and the "standard normal deviate" of no more than one occurrence in 1.67*N* trials (z). His formula is

$$C = M_g \sigma_g^{\ z} \qquad (3\text{-}1)$$

For example, if data for 1 year are available, if the 1-hour geometric mean concentration is 0.044 ppm, and if the 1-hour standard geometric deviation is 2.46, then the calculated maximum hourly concentration, expected to occur about once a year is

$$C_{\max(\text{hour})} = (0.044 \text{ ppm})\ (2.46)^{3.81} = 1.36 \text{ ppm},$$

since there are 8,760 hours in a year and the standard normal deviate corresponding to a probability of $\frac{1}{(8760)\ (1.67)}$ (or 0.0000685) is 3.81. Likewise, if M_g and σ_g are unchanged but the total period of observation is only 26 hours, then, since the standard normal deviate corresponding to a probability of $\frac{1}{(26)\ (1.67)}$ or (0.0231) is 1.99,

$$C_{\max(\text{hour})} = (0.044 \text{ ppm})\ (2.46)^{1.99} = 0.27 \text{ ppm}.$$

Or, in other words, this concentration is expected to be exceeded about once a day.

In a similar manner, it is possible also to calculate the minimum concentration for a given averaging time, as well as the concentrations at various percentiles, using the geometric mean and the standard geometric deviation for that averaging time. In addition, the geometric mean and the standard geometric deviation for one averaging time can be calculated from data obtained for other averaging times, although the accuracy of the results will depend on the number of air samples taken compared to the total number of samples that could have been taken if air sampling had been continuous. A set of 22 24-hour-duration random values were selected from the CAMP data by use of an NASN sampling schedule and were analyzed by Zimmer and Larsen.[1] For the 1-hour-averaging values, these investigators calculated a geometric mean of 0.054, as compared with 0.051, and a standard geometric deviation of 1.76, as compared with 1.94. Thus, a fairly good approximation of the year's hourly average frequency distribution could have been obtained with these 22 random samples.

Maxima calculated by Larsen's method are reasonable for averaging times of 1 hour or greater but tend to give overestimates for 5-minute periods; they do so for six of the eight cities in Table 3-1 (first column). The lower measured values may be partially due to the response times of the instruments. Table 3-2 compares the calculated maximum concentrations for six averaging times for the period from December 1, 1963, to December 1, 1964, with those actually observed in Chicago during the calendar year 1964. It is seen that the calculated value for averaging times as short as 5 minutes deviates significantly from the observed value. A plot of sulfur dioxide concentration data for Chicago from December 1, 1963, to December 1, 1964, is given in Figure 3-4. Averaging time is on a logarithmic scale, as is concentration in ppm, while frequency (percent) is given on a normal probability scale. The crosses and triangles in the figure denote observed values, while the lines represent calculated concentrations. For example, for an averaging time of 1 hour, 30 percent of the 1-hour-duration samples obtained during the year exceeded a concentration of about 0.2 ppm, and 0.1 percent of these samples showed a concentration in excess of 1 ppm. (These two points are marked with asterisks in the figure.) The 13 lines in the figure represent the 11 percentiles and the maximum and the minimum lines. In addition, several expected maximum concentration values are listed at the top of the figure. Thus the middle line represents the expected 50 percentile (or median) line, i.e., the concentration values which one would expect to be exceeded half the time at different averaging times. For example, for a 1-hour averaging time this concentration is about 0.12 ppm. At several points, percentile lines will coincide with maximum or minimum concentration lines. For instance, when the averaging time is 0.88 hour and the total time period is 1 year, the 0.01 percentile will correspond to the maximum, since there are 10,000 of these time-units in a year; similarly, the 99.99 percentile corresponds to the minimum.

Table 3-2.—COMPARISON OF CALCULATED AND OBSERVED MAXIMUM SULFUR DIOXIDE CONCENTRATIONS FOR VARIOUS AVERAGING TIMES IN 1964 IN CHICAGO.

Averaging time	Predicted maximum, ppm	Observed maximum, ppm
5 minutes	3.151	1.62
1 hour	1.646	1.12
8 hours	0.963	1.02
1 day	0.728	0.79
1 month	0.310	0.35
1 year	0.192	0.18

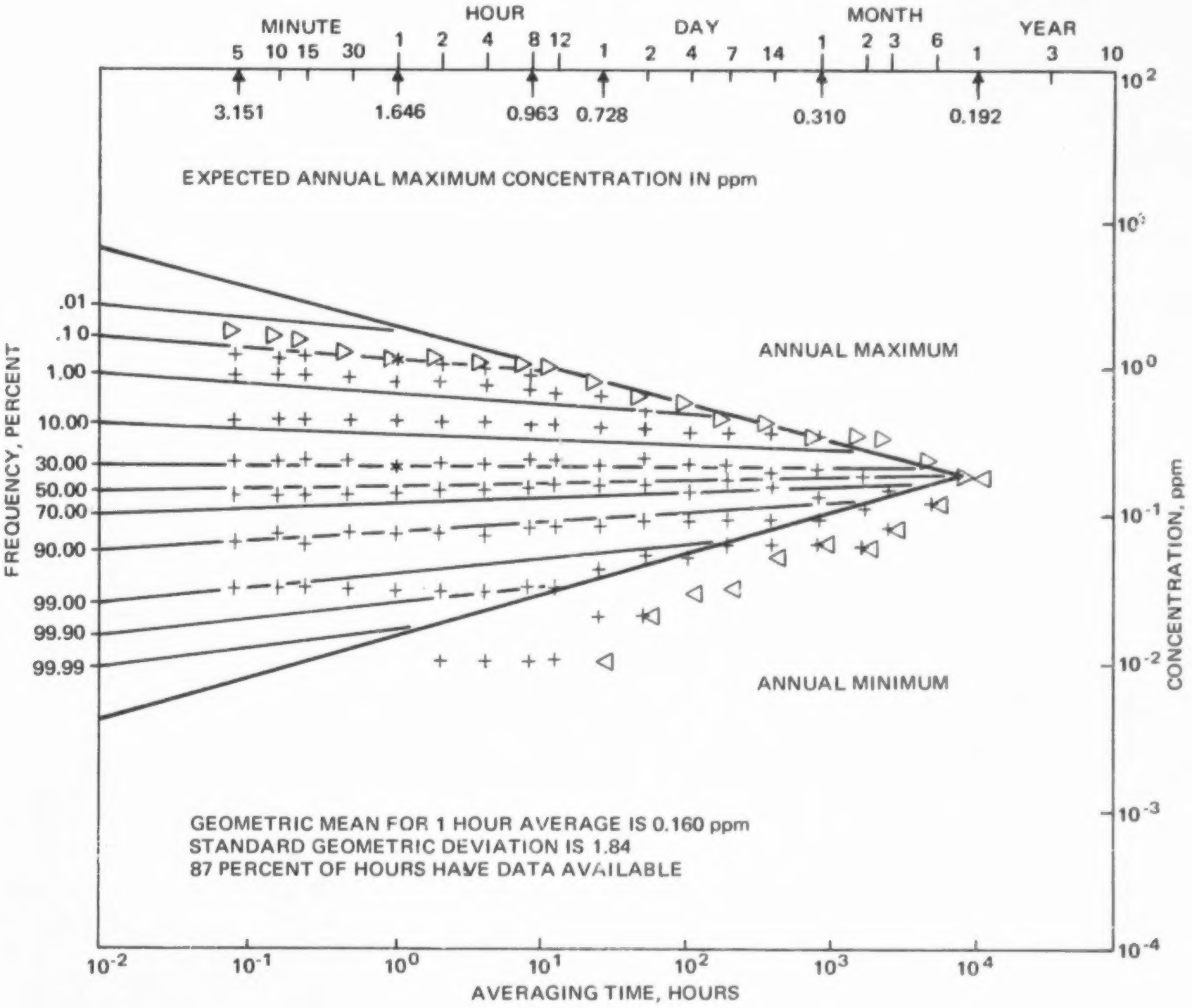

Figure 3-4. Concentration of Sulfur Dioxide in Chicago for Various Averaging Times and Frequencies, from December 1, 1963 to December 1, 1964.

For brief intervals, the concentration reached values as high as 1 ppm or 2 ppm, but for increasing averaging times, the upper and lower limits converge to a value near 0.2 ppm.

2. Concentrations Related to Point Sources

An industry which emits large quantities of sulfur dioxide is described as a point source. The area near such sources may experience high concentrations or immeasurably small values, depending on the prevailing winds, throughput differences, intermittent operations, intermittent pressure releases, and differences in velocity and temperature of emissions. Several studies of such situations have been reported.[4, 8]

a. Copper Smelter, Australia, 1962

Sullivan [9] reported that the maximum daily average concentrations were from 8 to 17 times the annual average concentration. At a station located 1.5 miles from the smelter, the hydrogen peroxide method gave a maximum daily average of 0.60 ppm, while the annual average was 0.036 ppm. A Thomas Autometer (conductometric) indicated peak concentrations from 1 ppm to 5 ppm on 30 occasions during a 6-month period and a maximum peak of 13.5 ppm.

b. Oil Refinery, 1965

Concentrations in excess of 0.5 ppm for 10 hours during 1 month with momentary peaks over 2 ppm in an area near an oil refinery were reported by Linzon.[7]

c. Smelter, Canada, 1958

A station located 8 miles from a smelter recorded an average concentration of 0.03 ppm during a 4-month study (May-August 1958). Measureable concentrations were present about 20 percent of the time, and concentrations above 0.25 ppm occurred 4 percent of the time.[10]

d. Smelter, Trail, Canada, 1931 to 1937

During the 6-month growing season, a station 15 miles south of the smelter recorded these data:

	1931	*1932*	*1933*	*1934*	*1935*	*1936*	*1937*
Average SO_2, ppm	0.032	0.007	0.008	0.012	0.023	0.022	0.013
Hours with $SO_2 > 0.25$ ppm	140	8.5	18	32.5	36	47	7

The concentrations are 6-month averages and the hours during which more than 0.25 ppm of sulfur dioxide was measured constitute from 0.2 percent to 3.5 percent of the total time. Maximum concentrations that lasted from 4 minutes to 54 minutes ranged from 0.48 ppm to 1.30 ppm or from 30 times to 160 times the respective 6-month averages.[8] The latter represents a considerably higher ratio than those which can be calculated from Table 3-1 and demonstrates the impact of point sources versus multiple sources.

e. Power Plant, 1966

Sulfur dioxide from a 1,000-megawatt power station burning 430 tons of 1.5 percent sulfur coal per hour was studied by Martin and Barber.[11] Sixteen sulfur dioxide recorders were spaced around a ring of radius about 3 miles to 4 miles centered on the station, i.e., near the zone of calculated maximum ground-level pollution. The maximum 3-minute concentration during the year was about 0.62 ppm, the maximum hourly average was about 0.47 ppm, the maximum daily average was 0.11 ppm, and the annual average was about 0.027 ppm.

f. Power Plant, Pennsylvania, 1961

One of the few studies of sulfur dioxide concentrations near a coal-fired power plant was made by McCaldin and Bye.[12] A Thomas Autometer ½ mile from this plant indicated an average concentration of 0.17 ppm from January to April (0.09 ppm by colormetric West-Gaeke) with a maximum hourly average of 2.9 ppm and a momentary peak concentration of 4.7 ppm.

C. SULFURIC ACID AND ITS RELATION TO SULFUR DIOXIDE

Measurements of sulfuric acid along with sulfur dioxide estimations are of interest because of the higher irritant potential of sulfuric acid, and because the ratio of the concentrations of the two pollutants found under various conditions helps in our understanding of the mechanism of the oxidation of sulfur dioxide to sulfuric acid in the atmosphere.

Coste [13, 14] made simultaneous measurements of sulfuric acid and sulfur dioxide in

London, England. Sulfur dioxide concentrations ranged from 0.13 ppm to 0.58 ppm (371 $\mu g/m^3$ to 1657 $\mu g/m^3$); sulfuric acid concentrations ranged from 4 $\mu g/m^3$ to 20 $\mu g/m^3$. The weight ratio of sulfuric acid to sulfur dioxide was 0.011 at the higher concentration of sulfur dioxide and 0.013 at the lower concentration. The highest ratio of sulfuric acid to sulfur dioxide was 0.023 on a misty day.

A maximum sulfur dioxide concentration of 1.47 ppm (4200 $\mu g/m^3$) in London during the period December 2 through December 5, 1957, was reported by Commins.[15] During the same period the maximum concentration of sulfuric acid was 222 $\mu g/m^3$. The ratio of maximum sulfuric acid to maximum sulfur dioxide was 0.053. Commins also reported that sulfuric acid could be as much as 10 percent of the total sulfur, corresponding to a weight ratio of sulfuric acid to sulfur dioxide of 0.167.

An extensive study of the simultaneous presence of sulfur dioxide and sulfuric acid mist in the air was made by Bushtueva.[16] [17] Between August 1953 and January 1954, she collected 198 paired samples for sulfur dioxide and sulfuric acid determination. The data are presented in Table 3-3. The sulfuric acid and sulfur dioxide were originally reported in mg/m^3 rather than in $\mu g/m^3$ as shown in the table. The concentrations of sulfur dioxide in ppm have been added for convenience of comparison with other data reported in ppm. These data show that as the sulfur dioxide concentration increases so does the sulfuric acid concentration, although at a slower rate.

Bushtueva also studied the effect of wind speed and relative humidity on the concentrations of sulfur dioxide and sulfuric acid. Both the sulfuric acid concentration and the ratio of sulfuric acid to sulfur dioxide were highest during periods of fog, and lowest during periods of precipitation. In the absence of precipitation, the ratio of sulfuric acid to sulfur dioxide increased from about 0.045 at 60 percent relative humidity to about 0.090 at 90 percent relative humidity, and to 0.15 at relative humidities above 91 percent. At wind speeds below about 4.5 miles per hour, the ratio of sulfuric acid to sulfur dioxide was 0.173, and at wind speeds greater than about 9 miles per hour, the ratio was only 0.068. Thus calm days, high humidity, and especially foggy weather are associated with high concentrations of sulfuric acid.

Although they did not study the relation of sulfuric acid to sulfur dioxide, Mader *et al.*[18] found that the sulfuric acid concentration at Los Angeles increased as the relative humidity increased.

Chaney [19] studied the relationship between sulfur dioxide (West-Gaeke measurement) and sulfuric acid in the Los Angeles area.

Table 3-3.—CONCENTRATION OF SULFUR DIOXIDE AND SULFURIC ACID MIST OBSERVED IN AIR [16] [17]

SO_2 concentrations		Number of tests	Average concentration			$H_2SO_4 : SO_2$ Weight ratio
			SO_2		H_2SO_4	
$\mu g/m^3$	ppm		ppm	$\mu g/m^3$	$\mu g/m^3$	
24-hour samples:						
25–100	0.009–0.035	6	0.01	30	12.6	0.420
101–250	0.035–0.088	18	0.06	176	19.6	0.112
251–500	0.088–0.175	38	0.14	387	20.0	0.051
501–750	0.176–0.263	20	0.23	663	31.0	0.045
751–1000	0.264–0.350	6	0.30	866	29.0	0.033
Over 1000	over 0.35	11	0.43	1220	43.0	0.035
Single samples:						
Up to 250	up to 0.088	15	0.05	128	17.5	0.137
251–750	0.088–0.263	8	0.15	428	41.6	0.097
Over 750	over 0.263	2	0.48	1380	326.0	0.235

During the time of the study, sulfur dioxide values were generally very low and sometimes there was no measurable sulfate. The weight ratios of sulfuric acid to sulfur dioxide ranged from 0.037 to 3.0 and the sulfate levels were relatively high compared to the sulfur dioxide concentrations. In the data of the National Air Surveillance Network, there is also a large amount of sulfate per unit of sulfur dioxide as measured by the West-Gaeke technique. In the strongly oxidizing atmosphere of Los Angeles, sulfur dioxide may be rather rapidly oxidized. On the other hand, the high ozone and nitrogen dioxide concentrations in Los Angeles may significantly interfere with the West-Gaeke procedure so that if the sulfamic acid modification is not used, values lower than actual sulfur dioxide concentrations may be recorded; consequently, oxidation of sulfur dioxide may be more apparent than real.

Thomas[20] used an automatic electroconductivity measuring instrument for the simultaneous measurement of sulfur dioxide and sulfuric acid mist concentrations during the winter of 1961 in Los Angeles. Concentrations of sulfur dioxide up to 0.21 ppm (600 $\mu g/m^3$) and of sulfuric acid up to 50 $\mu g/m^3$ were observed. Thomas originally reported the data in ppm; but to make them comparable with other data in this report they have been converted to $\mu g/m^3$ and are presented in Table 3-4. The weight ratios of sulfuric acid to sulfur dioxide lie between 0.032 and 0.246; these ratios are within the range of values reported by other investigators for other places.

Thomas' data indicate a nonlinear relationship between sulfur dioxide and sulfuric acid concentrations. Sulfuric acid increases as sulfur dioxide increases up to some critical value, depending upon the location. Beyond the critical value, sulfuric acid decreases as sulfur dioxide increases. In El Segundo, a maximum sulfuric acid concentration of about 25 $\mu g/m^3$ was observed when the sulfur dioxide concentration was between 0.15 ppm and 0.20 ppm (425 $\mu g/m^3$ to 570 $\mu g/m^3$),

Table 3-4.—CONCENTRATIONS OF SULFUR DIOXIDE AND SULFURIC ACID MIST OBSERVED IN 1961 IN AIR OVER LOS ANGELES AND ONE OF ITS SUBURBS, EL SEGUNDO[20]

Date	Sulfur dioxide		Sulfuric acid		$H_2SO_4 : SO_2$ Weight ratio
	ppm	$\mu g/m^3$	ppm	$\mu g/m^3$	
El Segundo					
26 Jan	0.065	185	0.0016	6.4	0.035
6 Feb	0.061	174	0.0031	12.4	0.071
22 Jan	0.062	177	0.0050	20.0	0.112
8 Feb	0.062	177	0.0054	21.6	0.112
10 Feb	0.055	158	0.0050	20.0	0.127
31 Jan	0.120	342	0.0046	18.4	0.054
11 Feb	0.102	291	0.0047	18.8	0.064
28 Jan	0.110	313	0.0055	22.0	0.070
15 Feb	0.125	356	0.0038	27.2	0.076
9 Feb	0.130	371	0.0075	30.0	0.081
30 Jan	0.205	584	0.0048	19.2	0.032
2 Feb	0.194	553	0.0098	39.2	0.071
Los Angeles:					
24 Mar	0.057	162	0.0048	19.2	0.118
21 Mar	0.067	191	0.0070	28.0	0.146
22 Mar	0.063	180	0.0072	28.8	0.160
10 Mar	0.057	162	0.0080	32.0	0.200
9 Mar	0.064	182	0.0092	36.8	0.201
13 Mar	0.065	185	0.0099	39.6	0.214
14 Mar	0.050	143	0.0084	35.2	0.246
22 Mar	0.122	348	0.0072	28.8	0.082
13 Mar	0.122	348	0.0126	50.4	0.145

whereas, in downtown Los Angeles, a maximum sulfuric acid concentration of about 30 $\mu g/m^3$ was observed when the sulfur dioxide concentration was between 0.05 ppm and 0.10 ppm (140 $\mu g/m^3$ to 280 $\mu g/m^3$).

During the past several years the National Air Surveillance Networks have taken simultaneous measurements of 24-hour average sulfur dioxide and suspended sulfate in various cities.[21-23] Unpublished analyses of these data show that correlation coefficients between sulfur dioxide and suspended sulfate (sulfuric acid and sulfate salts) range between 0.5 and 0.9. Manganese and iron in the suspended particulate matter, relative humidity, and temperature were studied as variables that might affect this correlation. As relative humidity and the concentration of metals increased, so did suspended sulfate. Temperature had no effect on suspended sulfate concentrations.

The data from field studies thus agree essentially with data from laboratory investigations; both show that sulfur dioxide can be oxidized to sulfuric acid or a salt of the acid in the atmosphere. The field studies show, from evidence taken in a number of geographical locations, that a relationship exists between sulfur dioxide and sulfuric acid concentrations in the air. The relationship is dependent partly upon the amount of moisture in the air, partly upon the time the sulfur contaminants have been in the atmosphere, the amount of catalytic particulate matter present in the air, the amount (intensity and duration) of sunlight, the amounts of hydrocarbons and oxides of nitrogen, and the amount of directly reactive and adsorptive materials in the air, as well as on the extent of recent precipitation.

D. ACIDITY OF RAINWATER AND PARTICULATE MATTER

Gorham[24] studied the acidity (pH) of rainfall in two cities of England and found that it was more strongly related to the chloride content than to the sulfate content. Such a study apparently has not been made in the United States; however, examination of some rainfall samples collected by the National Air Surveillance Network showed pH values around 3. The National Air Surveillance Network data indicate that the chloride content of suspended particulate matter is generally very low in atmospheres of the United States[25] except along coastal areas.

In another set of unpublished data collected by the National Air Pollution Control Administration, sulfate accounted for 21 percent of the variation in pH of solutions of the suspended particulate matter, with an increase of 12 $\mu g/m^3$ of sulfate reducing the pH by 1 unit. The pH values in both sets of data ranged from approximately 4 to 7.5. In the presence of substantial amounts of ammonia, calcium carbonate, hydroxide, or other alkaline material, most of the sulfate is in salt form. The similarity of the magnitude of atmospheric sulfuric acid concentrations, when compared with the magnitude of total sulfate concentrations, indicates that in some atmospheres a large part of the total sulfate may be sulfuric acid.[19-22 26]

Measurements of acidity of dustfall, though seldom reported, indicate that dustfall may be capable of providing a few pounds of hydrogen ions and a few hundred pounds of sulfate ions per acre per year.[27-29] The importance of this to soil management is difficult to evaluate because of the heterogeneity of soils, their generally great buffering capacity, and the probable large, though unknown, buffering capacity of the dustfall itself. An order of magnitude estimation of the effect of acidity in dustfall is that approximately twice as much lime might have to be applied to the soil in areas near large cities, as to the soil of rural areas. This assumes that the near-urban areas have average annual sulfate deposits of eight to twelve tons per square mile per month (300 to 450 pounds per acre per year). An approximately tenfold reduction in sulfate deposit would be required to reduce it to about the amount removed by plants and leaching.[30 31] Even though sulfur in some compounds is a major plant nutrient and is used as a fertilizer element under certain conditions, it would seem that uncontrolled deposition of sulfate from air pollution is undesirable.[32]

E. SUMMARY

Two major programs for the surveillance of atmospheric sulfur dioxide on a nationwide basis are operated by the U.S. Public Health Service and are (1) the National Air Surveillance Networks (NASN), in which 24-hour samples are obtained from about 100 sites 26 times a year, and (2) the Continuous Air Monitoring Project (CAMP), in which 5-minute average concentrations are recorded continuously in six large U.S. cities —Washington, Philadelphia, Cincinnati, Chicago, St. Louis, and Denver. The NASN program employs the colorimetric (i.e., West-Gaeke) method of analysis, while the CAMP program uses the electro-conductivity technique and recently has added continuous, colorimetric (West-Gaeke) SO_2 monitoring devices at all six locations.

Levels recorded in CAMP cities over the period from 1962 to 1967 show mean annual concentrations ranging from 0.01 ppm, in San Francisco, to 0.18 ppm, in Chicago, with the averages exceeding, for 1 percent of the time, a concentration range between 0.09 ppm and 0.68 ppm for the different cities. The NASN annual average concentrations ranged from 0.002 ppm, in Kansas City, Missouri, to 0.17 ppm, in New York City. The highest 24-hour average concentration was 0.38 ppm, also in New York City, while the lowest 24-hour avearges were below the minimum value detectable by the instruments—below approximately 0.001 ppm. Geographically, the highest values were recorded in the northeastern part of the United States, especially east of the Mississippi River and north of the Ohio River, where large quantities of high-sulfur fossil fuels are burned.

Extensive monitoring programs, such as those described above, yield large quantities of data. Information expressed in terms of average or maximum values does not give as complete a picture of air quality as does frequency distribution information, which shows the percentage of time that concentrations exceed specified levels. For a given averaging time, measurements of sulfur dioxide concentrations follow a log-normal frequency distribution. The two statistical parameters commonly used to describe this distribution are the geometric mean and the standard geometric deviation. CAMP data covering eight cities from 1962 to 1967 show that, as averaging times become shorter, higher maxima and lower minima are obtained. This happens because, for shorter intervals, instantaneous fluctuations have greater effect on the recorded values. The standard geometric deviation, which is an index of the deviation of the samples from their geometric mean, therefore is greater for shorter averaging periods.

Based on data from eight CAMP cities, the 1-hour maximum value for the year is expected to be about 10 to 20 times the annual average, corresponding to standard geometric deviations of 2 and 2.5, respectively. Thus, if the highest hourly average concentration for the year is 0.3 ppm, the annual mean would be about one-twentieth of this value, or 0.015 ppm, for a city with a standard geometric deviation of 2.5 and about one-tenth of this value, or 0.03 ppm, for a city with a standard geometric deviation of 2. Similarly, the 8-hour maximum ranges from about 6 to 10 times the annual mean; the one-day maximum ranges from 4 to 7 times the annual mean; and the maximum monthly average ranges from about 1.5 to 2 times the annual mean.

The CAMP data appear to be fairly representative of large U.S. metropolitan areas, but, for a city with a deviation outside these boundaries, a method exists to calculate the expected maximum concentrations, providing the averaging times are 1 hour or greater.[3] It is possible also to calculate the minimum concentration for a given averaging time, as well as the concentrations at various percentiles. These calculations make use of the geometric mean and the standard geometric deviation of samples obtained at a particular averaging time, and it is possible to derive the geometric mean and the standard geometric deviation for one averaging time from samples obtained at other averaging times. The accuracy of methods for calculating expected maxima, minima, or other concentrations from observed data for the same or for different averaging times depends on the number of air samples taken relative to the number of samples that could be taken if air sampling were continuous. It appears that a fairly good approximation of the frequency distribution of hourly averages over a year may be obtained with samples covering 24-hour periods on 22 randomly selected days.

Frequency data on the percent of the time that certain concentrations are exceeded usually necessitate the use of a computer but provide a very detailed description of air quality. For a given percentile, the concentrations measured over different averaging times usually vary. However, for the 30-percentile point, i.e., the value which is not exceeded for 70 percent of the time, the concentration appears to be about the same for all averaging times. Thus the 30-percentile value might be used as an indicator of the "general sulfur dioxide level" of a city, an index useful for ranking one city against another.

The ratio of the maximum sulfur dioxide concentration to the average value may be greater for measurements made near a single point source than for a city as a whole. For averaging periods from 4 to 54 minutes, for example, the maximum concentrations encountered near a point source were, respectively, 30 to 160 times the 6-month average value. These ratios are considerably higher than those calculated from the data for eight cities and 6 years shown in Table 3-1, and they demonstrate the kind of impact that a point source may have.

Hourly average concentrations of sulfur dioxide as high as 2.9 ppm have been measured ½ mile from a coal-fired power plant. In general, concentrations decrease as (1) the distance from sources increase, and (2) the averaging time is extended. When averaged over many hours or days, concentrations measured several miles from a large source may be only a few hundredths of a part per million. Near the point of discharge, power plants, oil refineries, smelters, and sulfuric acid production units may yield high ground-level sulfur dioxide concentrations.

Sulfur dioxide is partially oxidized to sulfuric acid in the atmosphere. The ratio between the two substances is dependent, among other things, on the oxidation rate, which in turn is dependent on the sunlight available and the concentration of moisture, catalysts, hydrocarbons, nitrogen oxides, and the quantity of directly reactive and adsorptive materials in the air. Recent precipitation also has an effect, as does the time various sulfur contaminants have been in the atmosphere.

F. REFERENCES

1. Zimmer, C. E. and Larsen, R. I. "Calculating Air Quality and its Control." J. Air Pollution Control Assoc., Vol. 15, pp. 565–572, Dec. 1965.
2. Brasser, L. J., Joosting, P. E., and van Zuilen, D. "Sulfur Dioxide—To What Level is it Acceptable?" Research Institute for Public Health Engineering, Report G–300, Delft, The Netherlands, July 1967.
3. Larsen, R. I. "A New Mathematical Model of Air Pollution Concentration, Averaging Time, and Frequency." (Presented at 61st Annual Meeting of the Air Pollution Control Association, St. Paul, Minnesota, June 23–27, 1968.)
4. "Concentrations of Volatile Sulfur Compounds in Atmospheric Air." Air Industrial Hygiene Foundation of America, Inc., Spec. Res. Bulls. 1 and 2, 1937, 1938.
5. Commins, B. T. and Waller, R. E. "Observations for a Ten-Year Study of Pollution at a Site in the City of London." Atmos. Environ., Vol. 1, pp. 49–68, 1967.
6. "Report of the International Joint Commission, United States and Canada, on the Pollution of the Atmosphere in the Detroit River Area." International Joint Commission (United States and Canada), Washington, D.C./Ottawa, Canada, p. 115.
7. Linzon, S. N. "Sulfur Dioxide Injury to Trees in the Vicinity of Petroleum Refineries." Forest Chronicle, Vol. 41, pp. 245–250, June 1965.
8. Katz, M. "Sulfur Dioxide in the Atmosphere of Industrial Areas." In: Effect of Sulfur Dioxide on Vegetation, National Research Council, Ottawa, Canada, 1939, pp. 14–50.
9. Sullivan, J. L. "The Nature and Extent of Pollution by Metallurgical Industries in Port Kembla." In: Air Pollution by Metallurgical Industries, Div. Occupational Health N. S. W., Dept. of Public Health, Sydney, Australia, Vol. 1, 1962, pp. 1–59.
10. Linzon, S. N. "The Influence of Smelter Fumes on the Growth of White Pine in the Sudbury Region." Joint Pub. Ontario Dept. Lands and Forests/Toronto Dept. Mines, 1958.
11. Martin, A. and Barber, F. R. "Investigations of Sulfur Dioxide Pollution Around a Modern Power Station." J. Inst. Fuel, Vol. 39, pp. 294–307, July 1966.
12. McCaldin, R. O. and Bye, W. E. "Air Pollution Appraisal, Seward and New Florence, Pennsylvania." U.S. Public Health Service, Robert A. Taft Sanitary Engineering Center, 1961.
13. Coste, J. H. "Investigation of Atmospheric Pollution." Cong. Intern. Quim. Pura Aplicada (Madrid), Vol. 6, pp. 274–287, 1934.
14. Coste, J. H. and Courtier, G. B. "Sulfuric Acid as a Disperse Phase in Town Air." Trans. Faraday Soc., Vol. 32, pp. 1198–1202, 1936.
15. Commins, B. T. "Determination of Particulate Acid in Town Air." Analyst, Vol. 88, pp. 364–367, May 1963.

16. Bushtueva, K. A. "Ratio of Sulfur Dioxide and Sulfuric Acid Aerosol in Atmospheric Air, in Relation to Meteorological Conditions." Gig. i Sanit., Vol. 11, pp. 11–13, 1954. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 4, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., August 1960, pp. 193–196.

17. Bushtueva, K. A. "The Determination of the Limit of Allowable Concentrations of Sulfur Acid in Atmospheric Air." In: Limits of Allowable Concentrations of Atmospheric Pollutants, Book 3, 1957, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., pp. 20–36.

18. Mader, P. P., Hamming, W. J., and Bellin, A. "Determination of Small Amounts of Sulfuric Acid in the Atmosphere." Anal. Chem., Vol. 22, pp. 1181–1183, Sept. 1950.

19. Chaney, A. L. "Investigations to Detect the Atmospheric Conversion of Sulfur Dioxide to Sulfur Trioxide." Am. Petrol. Inst., Vol. 38, pp. 306–312, 1958.

20. Thomas, M. D. "Sulfur Dioxide, Sulfuric Acid Aerosol and Visibility in Los Angeles." Int. J. Air Water Pollution, Vol. 6, pp. 443–454; Nov.-Dec. 1962.

21. "Air Quality Data (1962) National Air Sampling Network." U.S. Dept. of Health, Education, and Welfare, Public Health Service, 1964.

22. "Air Quality Data (1963) National Air Sampling Network." U.S. Dept of Health, Education, and Welfare, Public Health Service, 1965.

23. Perry, W. H. and Tabor, E. C. "National Air Sampling Network Measurement of SO_2 and NO_2." Arch. Environ. Health, Vol. 4, pp. 254–264, March 1962.

24. Gorham, E. "Atmospheric Pollution by Hydrochloric Acid." Quart. J. Roy. Meteorol. Soc., Vol. 84, pp. 274–276, July 1958.

25. "Air Pollution Measurements of the National Air Sampling Network. Vol. I. Analyses of Suspended Particulates, 1953–1957." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS-Pub-637, 1958.

26. "Air Pollution Measurements of the National Air Sampling Network. Vol. II. Analysis of Suspended Particulates, 1957–1961." U.S. Dept. of Health, Education, and Welfare, Public Health Service, PHS-Pub-978, 1962.

27. "Air Pollution in the El Paso, Texas Area. Report on a Two-Year Study under a Community Air Pollution Demonstration Project Grant." El Paso City-County Health Unit, 1959.

28. Katz, M. "Some Aspects of the Physical and Chemical Nature of Air Pollution." W.H.O. Monograph Series 46, Columbia Univ. Press, New York, 1961, pp. 97–158.

29. Junge, C. E. "Sulfur in the Atmosphere." J. Geophys. Res., Vol. 65, pp. 227–237, Jan. 1960.

30. Lyon, T. L., Buckman, H O., and Brady, N. O. "The Nature and Properties of Soils." 6th edition, Macmillan, New York, 1960, 567 pp.

31. Bertramson, B. R., Fried, M., and Tisdale, S. L. "Sulfur Studies of Indiana Soils and Crops." Soil Sci., Vol. 70, pp. 27–41, 1950.

32. Katz, M. "Sulfur Dioxide in the Atmosphere and its Relation to Plant Life." Ind. Eng. Chem., Vol. 41, pp. 2450–2465, Nov. 1949.

Chapter 4

EFFECTS OF SULFUR OXIDES IN THE ATMOSPHERE ON MATERIALS

Table of Contents

Page

A. INTRODUCTION 51

B. EFFECTS ON PAINTED SURFACES 51

C. DAMAGE TO METALS 51

D. EFFECTS ON BUILDING MATERIALS 54

E. EFFECTS ON TEXTILE FIBERS, DYES, AND MISCELLANEOUS MATERIALS 54

F. SUMMARY 55

G. REFERENCES 56

List of Figures

Figure

4–1 Relationship Between Corrosion of Mild Steel and Corresponding Mean Sulfur Dioxide Concentration for 3-, 6-, and 12-Month Exposures at Seven Chicago Sites (September 1963–1964) 53

Chapter 4

EFFECTS OF SULFUR OXIDES IN THE ATMOSPHERE ON MATERIALS

A. INTRODUCTION

In reviews of the effects of air pollution on materials, Yocom[1] and Burdick and Barkley[2] discuss the damage to metals, building materials, leather, paper, and textiles by the oxides of sulfur. Much of this damage is due to the conversion of sulfur oxides to highly reactive sulfuric acid. The combined action of sulfur oxides and particulate matter is discussed in the companion report *Air Quality Criteria for Particulate Matter.*

B. EFFECTS ON PAINTED SURFACES

Holbrow[3] found that the drying times of linseed, tung, and bodied dehydrated castor oil paint films exposed to 1 to 2 ppm SO_2 were increased by 50-100 percent. At higher concentrations, 7 to 10 ppm, drying was delayed by up to two to three days. Time of exposure to SO_2 was not mentioned. It may be assumed, however, that the exposure lasted until the paint films were dry. Oleoresinous and alkyd paints pigmented with titanium dioxide had both their touch- and hard-dry times increased substantially.

Based on these results Holbrow concluded that concentrations of SO_2 encountered in fogs or near industrial sites can increase the drying time and hardening time of certain kinds of paint systems. Some films become softer and others more brittle than those dried in the absence of SO_2—a factor likely to influence subsequent durability.

Holbrow[3] also found that when several types of paints (simple oil, oleoresinous or alkyd) were allowed to dry in clean air for 24 hours, then exposed to moisture and abnormally high levels of SO_2 (12,000 ppm or more; exposure time not specified), followed by exposure to warmth (temperature not stated) and moisture (quantity not indicated) for one hour, a significant reduction in gloss developed. Depending on the type of paint, the reduction in gloss, expressed as a percentage of the original value, ranged from 85 percent to 10 percent. Alone, SO_2 and moisture caused no more than a small reduction in gloss; it is only after exposure to further moisture and warmth that a large change occurred. It appeared that SO_2 rendered the films water sensitive. Control experiments omitting SO_2 showed no loss of gloss. Sensitivity to SO_2 is gradually lost the longer paint is allowed to dry prior to exposure.

The blueing of Brunswick green occurs when freshly applied paint is exposed to SO_2 levels of 0.2 ppm (duration of exposure not indicated) and subsequently exposed to warmth and moisture.[3]

Another troublesome paint defect is crystalline bloom which results when paints are exposed to SO_2, moisture, and ammonia. Bloom is due to the formation of very small crystals of ammonium sulfate. Calculated SO_2 levels of 0.1 ppm (duration of exposure not indicated) could cause moderate bloom.

C. DAMAGE TO METALS

Under normal conditions damage to metals by the oxides of sulfur increases with increasing relative humidity and temperature. Particulate matter in the air also contributes to the deleterious effects. Sulfur oxides generally accelerate corrosion by first being converted to sulfuric acid, either in the atmosphere itself or on metal surfaces. The corrosion products are mainly sulfate salts of the exposed metals.[4-7] In addition, atmospheric sulfuric acid can react with some suspended particles to form sulfate salts that also can accelerate corrosion of many metals.

Laboratory studies were conducted by Preston and Sanyal[8] using bare and varnished steel test panels, which were properly polished and degreased and then inoculated with finely divided particles of various dusts. The dusts were powdered oxides and chloride, sulfate, and chromate salts, and boiler and flue dusts like those commonly found in the atmosphere. The metal test panels were then exposed to atmospheres of pure clean air and air containing SO_2 (very low concentration but not specified) at various humidities, and the resulting corrosion was measured. Filiform corrosion, characterized by a filamental configuration, the primary phase in electrolytic corrosion, was noted in all cases. Corrosion at relative humidities below 70 percent is low, but it increases at higher humidities.[6, 8-11] In most of the cases in this study,[8] corrosion increased with humidity even in clean air. The addition of traces of SO_2 to the test atmospheres greatly increased the rate of corrosion in all instances.[8]

Atmospheres polluted with sulfur dioxide have been found to be among the most corrosive of all atmospheres studied, and to be even more corrosive than some marine atmospheres.[11, 12] A striking example is the almost fourfold reduction in the corrosion rate of zinc in Pittsburgh associated with a threefold reduction in sulfur dioxide, from annual levels of 0.15 ppm to 0.05 ppm, and a twofold reduction in dustfall in the period 1926 to 1960.[11, 13]

Schikorr[14] observed that steel panels exposed in Berlin during the winter months initially corroded five times faster than similar panels first exposed during the summer months. The particulate levels were much higher in winter and this resulted in the production of a greater abundance of corrosion nuclei. Nevertheless, Schikorr attributed this greater rate of corrosion primarily to the greater concentrations of gaseous combustion products, including sulfur oxides, in the air and to the longer periods of specimen wetting during the winter.

Foran *et al.*[15] and Gibbons[16, 17] found that, among several metals exposed to atmospheres ranging from rural to heavy industrial, carbon steels were most affected, followed in descending order by zinc, copper, aluminum, and stainless steel. Furthermore, they observed rather direct relationships between corrosion and oxides of sulfur pollution in several ambient atmospheres in which 2-year average lead peroxide candle sulfation rates ranged approximately from essentially 0 to 12 mg $SO_3/100$ cm^2-day. The increase in corrosion per unit increase in sulfation was greater at lower sulfation rates, e.g. of the maximum corrosion noted at 12 mg $SO_3/100$ cm^2-day, approximately one-half had occurred with steel and about one-fifth with zinc at 2 mg $SO_3/100$ cm^2-day. In terms of the measurements made by these investigators, a lead peroxide sulfation rate of 2 mg $SO_3/100$ cm^2-day is roughly equivalent to 0.08 ppm of sulfur dioxide.

Upham[18] reported on studies in St. Louis and Chicago where mild low-carbon steel panels were exposed to the atmosphere at a number of sites. It was assumed that meteorological conditions within one metropolitan area are sufficiently uniform that the air pollution level is the major site variable influencing observed corrosion rates, and concomitant measurements of the pollution levels at each site were made. In both cities, high correlations were found between corrosion rates, as measured by weight loss, and sulfur dioxide concentrations measured by the West-Gaeke method. Sulfation rates in St. Louis, measured by lead peroxide candles, also correlated well with weight loss due to corrosion. In St. Louis, except for one exceptionally polluted site, corrosion losses averaged 30 percent to 80 percent more then in nonurban locations.

Figure 4-1 shows the weight loss in 100-gram panels exposed in Chicago at seven sites for 3-, 6-, and 12-month periods and the corresponding mean concentration of sulfur dioxide measured at each site. Over the 12-month exposure period, the corrosion rate at the most corrosive site (mean SO_2 level of 0.12 ppm) was about 50 percent more than at least corrosive site (mean SO_2 level of 0.03 ppm). A regression analysis on the Chicago data relating corrosion, as measured by weight loss, to mean SO_2 concentrations resulted in the following regression equation:

$$y = 54.1\,s + 9.5$$

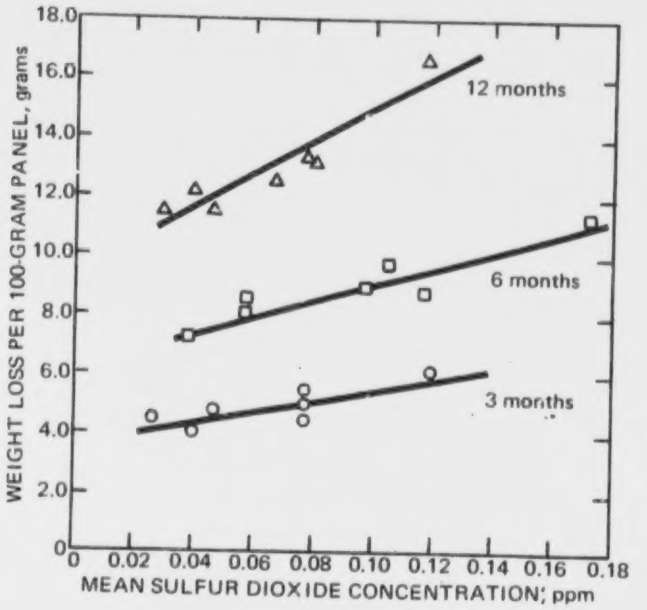

Figure 4-1. Relationship between corrosion of mild steel and corresponding mean sulfur dioxide concentration for 3-, 6-, and 12-month exposures at seven Chicago sites (Sept. 1963–1964).

where

y = corrosion weight loss in grams per hundred gram panel (4″ × 6″ × 0.035″ dimensions) and

s = mean SO_2 concentration in ppm.

The regression coefficient was statistically significant at the one-percent level. The linear relationship expressed by this equation would be valid only over the range of the observed data (i.e., from 0.03 to 0.12 ppm mean annual concentration) and in locatons with topography and climate similar to that of Chicago. The relationships were linear also in St. Louis, although the slope for the 3-month exposure period was greater than that in Chicago, presumably because the 3-month exposure took place during the winter months in St. Louis and during the autumn in Chicago. Differences between the two cities in terms of climate and exposure site configuration also may have been influential.

Suspended particulate levels were measured in Chicago at all seven sites with high-volume samplers, and these values correlated with corrosion rates. Since sulfur dioxide concentrations and particulate concentrations themselves tend to correlate, a covariance analysis was carried out to determine the relative influence of these two pollutants. This adjusted analysis indicated that the difference in corrosion rates at the various sites resulted primarily from differences in sulfur dioxide concentrations and not from differences in particulate levels. Thus, sulfur dioxide levels appeared to have the dominant influence on corrosion rates. Dustfall measurements made in St. Louis did not show a statistically significant correlation with corrosion rates.

The economic significance of metal corrosion has not been adequately investigated. In England, it has been estimated that about one-third of the annual replacement cost for steel rails is caused by air pollution.[19] Couy [20-22] investigated the effect of atmospheric corrosion on maintenance and economics of overhead line hardware and guy wires in Pittsburgh. The conditions of actual materials in use over the period roughly from 1920 to 1940 were determined for areas of severe and average pollution. Areas of severe pollution included valleys traversed by railways and areas in direct windline with industrial fumes. Formulae were developed to determine the amount of inspection required, the materials needed, and the costs involved for a particular situation. No direct cost estimates were made but it appeared that the life of the materials in the severely polluted areas was reduced to about two-thirds of that of the materials in the areas of average pollution. The average annual sulfur dioxide concentration during the period of the study was initially high (0.15 ppm) but had declined to approximately 0.05 ppm by 1940 (0.04 ppm in 1963).[13]

The effects of air pollution on electric contacts result in increased costs because of the losses associated with increased resistance of contacts having corrosion films and because more costly, less reactive, metals must be used. The use of gold instead of silver for contacts costs about 14.8 million dollars annually. If gold could be replaced by palladium, an annual savings of 8 million dollars would result, but palladium tarnishes in a sulfur dioxide atmosphere.[23]

D. EFFECTS ON BUILDING MATERIALS

There has been little recent investigation of effects of ambient concentrations of sulfur oxides on building materials. Early work[2 25–27] shows that sulfur oxides may be associated with damage to various building materials. Sulfur dioxide, in the presence of moisture is converted to sulfurous or sulfuric acids which are capable of attacking a wide variety of building materials, including limestone, marble, roofing slate, and mortar.[28–30] Any carbonate-containing stone is damaged by having the carbonate converted to relatively soluble sulfates, and then being leached away by rainwater.[28 30] Other types of stone, such as granite and certain sandstones, in which the grains are cemented together with materials containing no carbonate, are relatively unaffected by sulfur oxides in the atmosphere.[28]

Building stones erode to a greater or lesser degree according to their chemical composition. Softer building stones, such as the limestones and dolomites, are attacked more readily by the acids formed by the interaction of industrial smokes (even in small quantities) and moisture.[31] The dolomites contain both calcium and magnesium carbonates, the latter being readily soluble in an acid environment; the dolomitic stones are thus more vulnerable.[26 31 32] The calcium sulfate formed on the surface of masonry is about twice as bulky as the carbonate of the stone from which it was formed, so the stone appears to grow leprous.[31] Granites, gneiss, and many sandstones, which do not contain carbonates, and well-baked bricks, glazed bricks, and glazed tiles are attacked less readily by the sulfurous and sulfuric acids from the atmosphere. Additionally, serious disintegration of stone, caused in part by the expansion due to corrosion of iron tie rods, can occur.[26] Slates containing carbonates and the calcareous sandstones often used as roofing materials are also attacked by polluted acid atmospheres.[26] Decay occurs mainly on the undersides, especially between the laps, where moisture is held as a thin film.

Baines [25] reported that the expansive force, which developed from the crystallization of calcium and magnesium salts along the cleavage planes of stones in the buildings in various English cities, slowly opened up the old sealed vents where they could be attacked by atmospheric acid, and even lifted fragments of stone several tons in weight. On the British Houses of Parliament, which were approximately 90 years old when the survey was made, he found sulfate, which could only have been derived from the attack upon the stone by atmospheric acid, in cracks and fissures as much as 20 inches from the surface. He observed that the increase in the volume of crystallization of from 1 to 4.2 had thrown off great pieces of stone. Over 35 tons of stone were picked off portions of the building without using tools.

The deterioration of some of the finest monuments of antiquity and thousands of pieces of sculpture and carving in the open, on public buildings, and on cathedrals is a matter of increasing concern. The rate of decay has accelerated in recent years because of the increased pace of Twentieth Century industrialization and larger emissions of combustion products containing sulfur oxides and particulate matter. Cleopatra's Needle, the large stone obelisk moved from Alexandria, Egypt, to London, has suffered more deterioration in the damp, smoky acid atmosphere of London in 70 to 80 years than in the earlier 3,000 or more years of its history.

E. EFFECTS ON TEXTILE FIBERS, DYES, AND MISCELLANEOUS MATERIALS

The vulnerability of textile fabrics and furnishings to acid products of combustion, which are often sorbed on the particles emitted simultaneously by combustion sources, depends on the chemical composition of the textiles.[32] Cellulosic vegetable fibers, such as cotton, linen, hemp, jute, rayons, and synthetic nylons, are particularly sensitive to acid products of combustion such as sulfurous and sulfuric acids. After exposure to these acids, such fibers lose tensile strength.[33] Animal fibers, such as wool and furs, are more resistant to acid damage.[32]

Dyed fabrics containing certain classes of dyes are attacked by acid compounds often sorbed on atmospheric particles; the dye coloring is reduced or sometimes destroyed entirely. Color changes of dyed fabrics exposed without sunlight to the atmospheres of Los Angeles, Chicago, Phoenix, and Sarasota, Florida, were studied during the period October through December, 1961.[34] [35] For some dyes, maximum and severe fading occurred in Chicago, where sulfur dioxide concentrations were highest (NASN annual average about 0.09 ppm).[36] [37]

In controlled environment studies,[38] selected dye-fabric combinations were exposed to a variety of pollutants (in the absence of light) in order to assess dye fading characteristics. Auto exhaust showed no fading; clean air plus SO_2 (1 ppm) showed no fading; irradiated auto exhaust produced significant fading in some dyes; and irradiated auto exhaust plus SO_2 (1 ppm) gave rise to fading in additional dyes as well as more pronounced fading in those dyes that faded without SO_2. This shows the positive synergistic effect of SO_2, and again emphasizes that in many cases damage is a function of pollutants working together rather than by themselves.

Leather has a strong affinity for SO_2, which causes it to lose much of its strength and ultimately to disintegrate. The storage of valuable leather-bound books in city libraries can pose a serious problem. Book bindings stored in rooms with a free exchange of polluted air were found to deteriorate much more rapidly than those stored in confined spaces or inside of glass cases.[26]

Paper also absorbs SO_2 which is oxidized to sulfuric acid. The sulfuric acid causes discoloration and renders the paper brittle and fragile.[26] Exposures to SO_2 (2-9 ppm) for 10 days resulted in embrittlement and a decrease in folding resistance of both book and writing paper.[2]

F. SUMMARY

Although more quantitative data presenting dose-response relationships are needed, sufficient evidence exists to conclude that atmospheres polluted with oxides of sulfur directly and indirectly attack and damage a wide range of materials and property. Much of this damage is due to the conversion of sulfur oxides to highly reactive sulfuric acid.

In the laboratory, steel test panels dusted with particles commonly found in polluted atmospheres (e.g., powdered oxides, boiler and flue dusts, and chloride, sulfate, and chromate salts) corroded at a low rate in clean air at relative humidities below 70 percent. The corrosion rate increased at humidities higher than 70 percent, and it greatly increased when traces of sulfur dioxide were added to the laboratory air.

Corrosion rates of most metals and especially iron, steel, and zinc are accelerated by sulfur dioxide-polluted environments. Particulate matter, high humidity, and temperature also play an important synergistic part in this corrosion reaction, Atmospheric corrosion studies show increased corrosion rates in industrial areas where air pollution levels, including sulfur dioxide and related pollutants, are higher. Further, corrosion rates are higher in the fall and winter seasons when particulate and sulfur oxides pollution is more severe due to a greater consumption of fuel for heating. Depending on the kind of metal exposed as well as location and duration of exposure, corrosion rates were 1-½ to 5 times greater in polluted atmospheres than in rural environments. In Pittsburgh, from 1926 to 1960, when annual sulfur dioxide levels were reduced from 0.15 ppm to 0.05 ppm, zinc corrosion rates exhibited a fourfold reduction. In Chicago and St. Louis, where steel panels were exposed at a number of sites, high correlations were found in each city between corrosion rates, as measured by weight loss, and sulfur dioxide concentrations measured by the West-Gaeke method. In St. Louis, except for one exceptionally polluted site, corrosion losses averaged 30 percent to 80 percent more than in nonurban locations. Over a 12-month exposure period in Chicago, the corrosion rate at the most corrosive site (mean SO_2 level of 0.12 ppm) was about 50 percent more than at the least corrosive site (mean SO_2 level of 0.03 ppm). These correlations were statistically significant at the one-percent level. Sulfation rates in St. Louis, measured by lead peroxide candle, also correlated well with weight loss due to corrosion. Although suspended particulate levels measured in Chicago with high-volume samplers correlated with corrosion rates, a covariance analysis indicated that sulfur dioxide concentrations were the dominant influence on corrosion. Measurements of dustfall in St. Louis did not correlate significantly with corrosion rates. Based on these data, it appears that considerable corrosion may take place (i.e., from 11 percent to 17 percent weight loss in steel panels) at annual sulfur dioxide concentrations in the range of 0.03 ppm to 0.12 ppm, and although high particulate levels tend to accompany high sulfur dioxide levels, the sulfur dioxide concentration has a greater influence on the degree of corrosion that takes place.

Qualitative examples of effects resulting from corrosion by sulfur dioxide include:

1. a one-third reduction in the life of overhead power line hardware and guy wires;
2. the necessary use of more expensive, less corrodible metals such as gold in some electrical contacts; and
3. one-third of the annual damage to steel rails in England.

Atmospheres containing oxides of sulfur also attack and damage a wide variety of building materials—limestone, marble, roofing slate, and mortar—as well as statuary and other works of art, causing discoloration and actual physical deterioration. Certain textile fibers—such as cotton, rayon, and nylon—are also harmed by atmospheric sulfur oxides, and in the presence of other pollutants, fading of dyed fabrics may occur. Severe fading was noted for some dyes in fabrics exposed in Chicago, where annual average sulfur dioxide levels were 0.09 ppm. Leather exposed to sulfur dioxide may lose much of its strength while paper becomes discolored and brittle.

Concentrations of 1 ppm SO_2 can increase the drying time of some oil-based paints by 50 percent to 100 percent. Some films become softer and others more brittle, a factor likely to influence subsequent durability. Sulfur dioxide also appears to render some paint films water sensitive, resulting in reduced gloss. Under certain conditions, SO_2 levels of 0.1 ppm to 0.2 ppm can cause the blueing of Brunswick green, and in the presence of ammonia produce a troublesome defect called crystalline bloom, due to the formation of very small ammonium sulfate crystals.

G. REFERENCES

1. Yocum, J. E. and McCaldin, R. O. "Effects of Air Pollution on Materials and the Economy." In: Air Pollution, 2nd edition, Vol. I, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 617–654.
2. Burdick, L. R. and Barkley, J. F. "Effect of Sulfur Compounds in the Air on Various Materials." U.S. Bureau of Mines, Information Circular 7064, April 1939.
3. Holbrow, G. L. "Atmospheric Pollution: Its Measurement and Some Effects on Paint." J. Oil Colour Chem. Assoc., Vol. 45, pp. 701–718, Oct. 1962.
4. Serada, P. J. "Atmospheric Factors Affecting the Corrosion of Steel." Ind. Eng. Chem., Vol. 2, pp. 157–160, Feb. 1960.
5. Greenblatt, J. H. and Pearlman, R. "The Influence of Atmospheric Contaminants on the Corrosion of Steel." Chem. Canada, Vol. 14, pp. 21–23, Nov. 1962.
6. Vernon, W. H. J. "The Corrosion of Metals, Lecture I." J. Roy. Soc. Arts, pp. 578–610, July 1, 1949.
7. Sanyal, B. and Bhardwar, D. V. "The Corrosion of Metals in Synthetic Atmospheres Containing Sulphur Dioxide." J. Sci. Ind. Res. (New Delhi), Vol. 18A, pp. 69–74, Feb. 1959.
8. Preston, R. St. J. and Sanyal, B. "Atmospheric Corrosion by Nuclei." J. Appl. Chem., Vol. 6, pp. 26–44, Jan. 1956.
9. Vernon, W. H. J. "A Laboratory Study of the Atmospheric Corrosion of Metals, Parts II and III." Trans. Faraday Soc., Vol. 31, pp. 1668–1700, Dec. 1935.
10. Vernon, W. H. J. "The Atmospheric Corrosion of Metals." Soc. Chem. Ind. (Chem. Eng. Group), Prof., Vol. 19, pp. 14–22, 1937.
11. Tice, E. A. "Effects of Air Pollution on the Atmospheric Corrosion Behavior of Some Metals and Alloys." J. Air Pollution Control Assoc., Vol. 12, pp. 553–559, Dec. 1962.
12. "Symposium on Atmospheric Corrosion of Non-Ferrous Metals." Spec. Tech. Pub. 175. (Presented at the 58th Annual Meeting, American Society for Testing Materials, Atlantic City, N. J., June 29, 1955.)
13. Anderson, D. M., Lieben, J., and Sussman, V. H. "Pure Air for Pennsylvania." Pennsylvania Dept. of Health and U.S. Dept. of Health, Education, and Welfare, Public Health Service, Nov. 1961.
14. Schikorr, G. "The Atmospheric Rusting of Iron." Korrosion and Metallschutz., pp. 305–313, Sept. 17, 1941.
15. Foran, M. R., Gibbons, E. V., and Wellington, J. R., "The Measurement of Atmospheric Sulphur Dioxide and Chlorides." Chem. Canada, Vol. 10, pp. 33–41, May 1958.

16. Gibbons, E. V. "Atmospheric Corrosion Testing of Metals in Canada." Corrosion, Vol. 17, pp. 318–320, 1961.
17. Gibbons, E. V. "The Corrosion Behavior of the Major Architectural and Structural Metals in Canadian Atmospheres. Summary of Two-Year Results." National Research Council, Ottawa, Canada, Feb. 25, 1959.
18. Upham, J. B. "Atmospheric Corrosion Studies in Two Metropolitan Areas." J. Air Pollution Control Assoc., Vol. 17, pp. 398–402, June 1967.
19. "Committee on Air Pollution, Beaver, H., Chairman, Report." Her Majesty's Stationery Office, London, 1954.
20. Couy, C. J. "Effect of Atmospheric Corrosion on Maintenance and Economics of Overhead Line Hardware and Guy Strand, Part 1." Corrosion (Natl. Assoc. Corr. Eng.), Vol. 4, pp. 133–140, April 1948.
21. Couy, C J. "Effect of Atmospheric Corrosion on Maintenance and Economics of Overhead Line Hardware and Guy Strand, Part 2." Corrosion (Natl. Assoc. Corr. Eng.), Vol. 4, pp. 207–218, May 1948.
22. Couy, C. J. "Effect of Atmospheric Corrosion on Maintenance and Economics of Overhead Line Hardware and Guy Strand, Part 3." Corrosion (Natl. Assoc. Corr. Eng.), Vol. 4, pp. 287–303, June 1948.
23. Gilbert, P. T. "The Protection of Steel Against Atmospheric Corrosion by Metallic Coatings." Ind. Chem., Belge., Vol. 19, pp. 405–415, Sept. 1963.
24. Antler, M. and Gilbert, J. "Effects of Air Pollution on Electric Contacts." J. Air Pollution Control Assoc., Vol. 13, pp. 405–415, Sept. 1963.
25. Baines, F. "Examination into Effects of Air Pollution on Building-Stones, etc." National Smoke Abatement Society, Manchester, England, 1934.
26. Parker, A. "The Destructive Effects of Air Pollution on Materials." In: Proceedings, 1955 Annual Conference, National Smoke Abatement Society, London, 1955, pp. 3–15. (Presented at Sixth Des Voeux Memorial Lecture.)
27. McBurney, J. W. "Effect of the Atmosphere on Masonry and Related Materials." In: Symposium on Some Approaches to Durability in Structures, Boston, Massachusetts, June 23, 1958, Spec. Tech. Pub. 236, pp. 45–52.
28. Yocum, J. E. "The Deterioration of Materials in Polluted Atmospheres." J. Air Pollution Control Assoc., *8*(4):203–208, Nov. 1958.
29. Benner, R. C., IV. "The Effect of Smoke on Stone." In: Papers on the Effect of Smoke on Building Materials, University of Pittsburgh, Mellon Institute of Industrial Research and School of Specific Industries, Bulletin 6, 1913.
30. Turner, T. H. "Damage to Structures by Atmospheric Pollution." Smokeless Air, Vol. 23, pp. 22–26, Autumn 1952.
31. Regan, C. J. "A Chadwick Lecture on Air Pollution." Smokeless Air, Vol. 88, pp. 67–76, 1953.
32. Petrie, T. C. "Smoke and the Curtains." Smokeless Air, Vol. 18, pp. 62–64, Summer 1948.
33. Waller, R. E. "Acid Droplets in Urban Air." Int. J. Air Water Pollution, Vol. 7, pp. 773–778, 1963.
34. Salvin, V. S. "Effect of Air Pollutants on Dyed Fabrics." J. Air Pollution Control Assoc., Vol. 13, pp. 416–422, Sept. 1963.
35. Salvin, V. S. "Relation of Atmospheric Contaminants and Ozone to Lightfastness." Am. Dyestuff Rep., Vol. 53, pp. 33–41, Jan. 6, 1964.
36. "Air Quality Data (1962) National Air Sampling Network." U.S. Dept. of Health, Education, and Welfare, Public Health Service, 1964.
37. "Air Quality Data (1963) National Air Sampling Network." U.S. Dept. of Health, Education, and Welfare, Public Health Service, 1965.
38. Ajax, R. L., Conlee, C. J., and Upham, J. B. "The Effects of Air Pollution on the Fading of Dyed Fabrics." J. Air Pollution Control Assoc., Vol. 17, pp. 220–224, April 1967.

BLANK PAGE

Chapter 5

EFFECTS OF SULFUR OXIDES IN THE ATMOSPHERE ON VEGETATION

Table of Contents

Page
A. INTRODUCTION 61
B. SYMPTOMS IN VEGETATION OF EXPOSURE TO SULFUR DIOXIDE 61
  1. Acute Injury 61
  2. Chronic Injury 62
  3. Physiological Effects 62
  4. Mechanism of Injury 63
C. SUSCEPTIBILITY OF VEGETATION TO SULFUR DIOXIDE 63
D. FACTORS AFFECTING PHYTOTOXICITY 65
  1. Temperature 65
  2. Relative Humidity 66
  3. Soil Moisture 66
  4. Light Intensity 66
  5. Nutrient Supply 66
  6. Age of Plant Tissue 66
E. SYNERGISTIC EFFECTS OF SULFUR DIOXIDE AND OZONE 66
F. EFFECTS OF SULFURIC ACID MIST ON VEGETATION 66
G. SUMMARY 67
H. REFERENCES 68

List of Figures

Figure
5–1. Curves Showing Concentrations of Sulfur Dioxide Which Will Produce Three Different Stages of Acute Alfalfa Leaf Damage in Different Time Periods under Conditions of Maximum Absorption 64

Chapter 5

EFFECTS OF SULFUR OXIDES IN THE ATMOSPHERE ON VEGETATION

A. INTRODUCTION

Extensive experiments and observations have been made for over one hundred years on the effects of sulfur dioxide on vegetation by investigators in all parts of the world. Substantial reviews of vegetation injury from the oxides of sulfur have been made by Thomas,[1–4] Sheffer and Hedgcock,[5] Brandt and Heck,[6] and Katz and McCallum,[7] and Daines.[8] Summaries of acute effects on a large number of plant species susceptible to sulfur dioxide injury are given by Wood [9] and in the Environmental Biology Volume of Biological Handbooks.[10] Although Thomas [4] stated that the effects of sulfur dioxide on plants are fairly well understood, there are still some points of unresolved controversy in the literature. The response of plants to sulfur dioxide presents a many faceted problem [8] and involves the interactions of plant characteristics, environmental conditions, and dosage of the toxicant.

According to Katz,[11] one of the first descriptions of sulfur dioxide injury to plants was by Schroeder and Ruess in 1883. Descriptions of injury have appeared in numerous publications since that time.[6–8 12–14] Two general types of injury are produced by sulfur dioxide, acute and chronic.[1 6–8] Any overall effect on growth and production is apparently associated with some degree of acute injury from sulfite or sulfurous acid or to chronic injury resulting from accumulation of sulfates.[14]

B. SYMPTOMS IN VEGETATION OF EXPOSURE TO SULFUR DIOXIDE

1. Acute Injury

Acute injury results from the rapid absorption of toxic concentrations of sulfur dioxide.[14] Immediately after exposure, tissues in sharply defined marginal and intercostal area take on a dull water-soaked appearance.[1] These areas subsequently dry out and bleach to an ivory color. On some species the lesions finally become brown to reddish-brown.[4 6 15 16] A sharp line usually separates the injured lesion from surrounding, apparently healthy, leaf tissue. Injury seldom extends across the veins of broad-leafed plants unless the injury is severe.[2]

Acute injury in pine usually occurs in bands on the tips of needles with the injured area taking on a red-brown color.[7] The injured area changes from the usual dark green color to a lighter green, then definite areas turn yellow-brown and finally red-brown, giving the banded appearance. The discoloration in conifers may involve the whole needle or limited areas of any portion. Abscission may follow after some interval and the affected trees are often deficient in needles.

The basic bleached and collapsed blotches described on broad-leafed plants are, however, also typical on grass foliage.[17 18] The final bleached pattern between the parallel veins of grass leaves gives a streaked effect. Often only the principal vein or "midrib" remains intact. The collapsed lesion generally extends uniformly though the entire thickness of the leaf blade.

Holmes *et al.*[12] found that barley, growing in the field, was severely injured by fumigations with 5 ppm sulfur dioxide administered one hour a day on six successive days. If the same total dosage was applied for much shorter intervals during the six days, there was much less injury. Apparently injury could be repaired if sufficient periods free of toxicant were provided. It was also reported that injury to very young barley plants did not reduce final yield but that injury late in the life of the plants caused significant reduction in yield. No yield reduction occurred unless visible damage symptoms were detected on the foliage.

2. Chronic Injury

Sublethal concentrations of sulfur dioxide may require several days or weeks to cause the development of the gradual yellowing or chlorotic symptom of chronic injury.[2] The slow fading of green color over a period of several days suggests that the chlorophyll-making mechanism is being destroyed and that the green pigment cannot be replenished. If only a few cells in an area are affected, the area may become chlorotic or brownish-red. A large amount of sulfate is found in leaves with chronic symptoms,[14] whereas leaves which are acutely injured show only a small increase in sulfate content. Large quantities of sulfate may be accumulated in leaf tissue without causing injury.[14 27] Yet, if excessive amounts are absorbed rapidly, acute injury results.

Sulfur dioxide is very soluble in water and is absorbed by leaves. If absorbed slowly it is oxidized or perhaps reduced to a small extent.[14] The sulfuric acid resulting from oxidation and hydrolysis in the leaf tissue is rapidly metabolized by organic bases. Acids formed in the leaf tissue may reduce the buffer capacity without changing pH and when the buffering capacity is exceeded, chlorosis results. A recent report [19] indicates that the reduction of sulfur dioxide in the leaf tissue may have more significance than previously proposed by Thomas,[14] since considerable hydrogen sulfide was given off by tomato plants during, and for a time after, fumigation with sulfur dioxide.

3. Physiological Effects

The "invisible injury" theory suggesting that suppression of growth and yield may occur because of long-term low concentration exposure to sulfur dioxide, even if visible symptoms of foliage injury do not develop, was proposed by Stoklasa.[20] According to Setterstrom,[21] this theory was supported by several investigators, including Wieler, Bredemann, Janson, and Bleasdale. Many investigators accept the thesis that growth and yield are not affected unless visible symptoms of injury occur on the foliage.[1 2 6 22–24] There is a straight-line relationship between yield of alfalfa and either the total area destroyed by acute symptoms or the area covered by the chlorotic symptom.[25] The amount of yield reduction produced by sulfur dioxide symptoms can be duplicated by clipping a comparable amount of leaf area from healthy plants.[16] Destruction of portions of the leaf area and accompanying defoliation not only reduces yield of the current crop but may adversely affect the next one or two clippings.

Reduction in growth of pine trees in the vicinity of sulfur dioxide sources has been reported.[7 26 27] This growth reduction was evidently due to heavy defoliation and destruction of portions of needles during prolonged exposure to levels of sulfur dioxide sufficient to kill cells and tissue or to build sulfate concentrations to a level which destroyed chlorophyll. Needles of many conifers are normally retained for three or more years and accumulate sulfate from continued low concentrations of sulfur dioxide. Needle drop is stimulated by high levels of sulfates, and trees growing near sulfur dioxide sources often have very sparse foliage.[27]

Defoliation of citrus trees exposed to sulfur dioxide has been observed in Japan.[28] Defoliation increased with increasing concentrations, and increased sulfur content of foliage coincided with the rate of defoliation. It was concluded that leaf analyses for sulfur content may be useful for evaluating chronic injury, but such analyses could not be used for evaluating acute injury. A lower rate of absorption may produce symptoms of chlorosis and reduce CO_2 exchange, causing temporary disruption of photosynthesis and respiration. The rate of carbon dioxide exchange returns to normal soon after the toxicant is removed if visible symptoms do not develop. At very low concentrations, the sulfur oxides may be utilized by plants deficient in sulfur causing stimulation of growth and production.[14 21 24] Bleasdale,[29] in a study conducted near Manchester, England, found that yields of rye grass grown in unfiltered air were significantly lower than similar plants grown in filtered air. The SO_2 levels in the unfiltered air ranged from 0.01 ppm to 0.06 ppm with exposure periods varying from 46 to 81 days.

No visible symptoms were observed on the plants exposed to SO_2; it is not known whether other gaseous pollutants were present.

4. Mechanism of Injury

The mechanism by which sulfur dioxide causes injury to leaf tissue is not thoroughly understood, although a number of suggestions have been made. The acute symptoms are generally attributed to excessive sulfite or sulfurous acid in the tissue.[1,14] With a high rate of absorption, sulfite is thought to accumulate; sulfurous acid is then formed and subsequently attacks the cells. Sulfur dioxide enters the leaf through the stomata and is thought to be oxidized, perhaps by oxygen, a by-product of photosynthesis.[23] Sulfuric acid is then formed and reacts with organic bases.[11] The resulting sulfates are apparently translocated with the transpiration stream and deposited at the tip or edges of the leaf. Haselhoff [30] suggested that sulfur dioxide attaches itself to aldehydes and sugars in the leaf and as these products degrade, sulfurous or sulfuric acid is formed and injury results. Dorries [31] reasoned that local acidity from the sulfur dioxide split magnesium from the chlorophyll molecule and formed pheophytin. Noack [32] suggested that iron in the chloroplasts was inactivated by sulfur dioxide, causing interference with the assimilation of organic compounds by the plant.

C. SUSCEPTIBILITY OF VEGETATION TO SULFUR DIOXIDE

Different species, different varieties within a plant species, and even individuals within a plant variety vary considerably in their susceptibility to sulfur dioxide.[6,8,14] Thomas [2–4] tabulates many plants according to their sensitivity to sulfur dioxide. Since alfalfa is one of the most sensitive plants, it was used as a reference species and all other species and varieties were compared with it. The order of sensitivity was calculated by Thomas [25] from unpublished data of O'Gara on resistance factors for over 300 plants. According to Thomas,[25] O'Gara's method of determining resistance consisted of fumigating the plants for one hour with a measured concentration of sulfur dioxide just sufficient to cause traces of injury. A corrected threshold concentration for injury was calculated for 100 percent relative humidity. This calculated value was then divided by 1.25 ppm, which was the concentration required for incipient marking on alfalfa in the 1-hour period. The ratings range from alfalfa, barley, endive, and cotton, the most sensitive, with ratings of 1, through celery, citrus, and cantaloupe, with ratings of about 7, to privet leaves, the most resistant with a rating of 15.

O'Gara's unpublished data and his fundamental equation for the "Law of gas action on the plant cell"[33] have been used extensively by Thomas [1,3,4,12,23] to calculate relative sensitivity of various plants to sulfur dioxide fumigation and to determine the effect on yield. O'Gara's formula was:

$$t(C - C_o) = K \qquad (5\text{-}1)$$

where t is time (hrs) through which the gas acts to produce a certain effect,

C is the concentration (ppm) of the gas,

C_o is the threshold concentration (ppm) of the gas for injury to the plant, and

K is a constant.

According to Thomas [25] the amount of injury caused by a given quantity of sulfur dioxide varies with the rate of absorption; that is, a given amount of gas, absorbed in a short time period, will cause more leaf destruction than if the absorption were to occur over a longer period. General time-concentration-absorption equations for data obtained from fumigations of alfalfa are proposed. By applying these equations to data from numerous fumigations, it was possible to calculate the dosage required to produce any observed degree of injury under conditions of maximum sensitivity. For example, equations representing incipient marking, 50 percent leaf destruction and 100 percent leaf destruction, under conditions of maximum sensitivity were:

$tC = 0.94 + 0.24t$ (traces of leaf destruction appear),

$tC = 2.1 + 1.4t$ (50 percent leaf destruction occurs), and

$tC = 3.2 + 2.6t$ (100 percent leaf destruction occurs).

Injury thus begins after four hours of exposure to 0.45 ppm, etc. Curves constructed from these calculations are plotted in Figure 5–1. Guderian *et al.*[32] did not believe that the O'Gara equation fitted their observations and suggested that an exponential equation of the form

$$t = ke^{-a}(C-r) \tag{5–2}$$

where *k*, *a*, and *r* are parameters varying with species and degree of injury, best described their data.

An apparatus was developed by Spierings[35] whereby susceptibility of trees and shrubs to air pollutants could be determined under natural conditions without disruption of prevailing climates. Fumigation experiments with sulfur dioxide showed that apple and pear varieties were the most susceptible of the fruit trees and that mountain-ash was one of the most susceptible of the deciduous forest trees. Six-hour exposures of the fruit varieties to 0.48 ppm produced injury, whereas injury to the mountain-ash was produced by three-hour exposures to 0.54 ppm of sulfur dioxide.

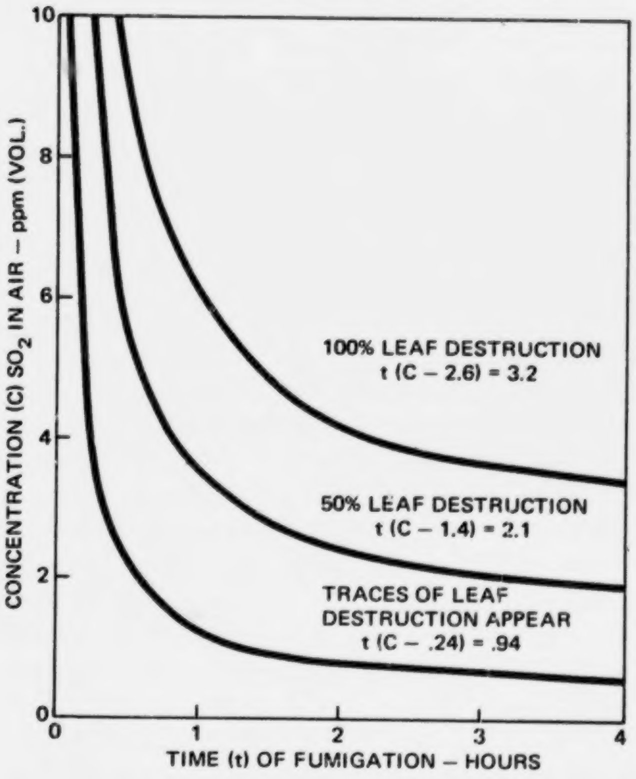

Figure 5-1. Curves Showing Concentrations of Sulfur Dioxide Which Will Produce Three Different Degrees of Acute Alfalfa Leaf Damage in Different Time Periods Under Conditions of Maximum Absorption.

These curves from experimental data may be used to estimate the extent of damage to alfalfa at any sulfur dioxide concentration and exposure time.

Sheffer and Hedgcock[5] reported injury to ponderosa pine, western larch, Pacific ninebark, and creambush rockspirea during an exposure to 0.5 ppm for 7 hours. They concluded that the lowest concentration of sulfur dioxide that injures conifers is 0.25 ppm. Alfalfa is generally considered to be one of the most sensitive plants, and Thomas[16] reported that photosynthesis or respiration by alfalfa plants was not affected by concentrations up to 0.3 ppm unless the exposure was so long that sulfate injury occurred. Katz and McCallum[7] reported that larch, one of the most sensitive conifers, developed slight symptoms from 0.3 ppm sulfur dioxide in an 8-hour exposure. They concluded that if concentrations higher than 0.3 ppm to 0.5 ppm do not occur and if the duration of concentration at the 0.3 ppm level is short in a single gas exposure, plant damage should not occur.

In the vicinity of the smelter at Trail, British Columbia, which in 1929 emitted an average of 18,600 tons of sulfur dioxide per month, plant injury was noted as far as 52 miles south of the smelter.[26] Three zones of injury were delineated on the basis of the percentage injury to ponderosa pine, Douglas fir, and forest shrubs: in Zone 1 there was 60 percent to 100 percent injury; in Zone 2, 30 percent to 60 percent injury; in Zone 3, 1 percent to 30 percent injury. Zone 1, in which injury was acute, extended about 30 miles south of the smelter in a river valley; Zone 3, at higher elevations, extended 52 miles south of the smelter and contained trees with relatively slight markings and trees suffering from slow but progressive deterioration. After sulfur dioxide control devices were installed, measurements in 1934 and again in 1935[26] showed that the injury to broadleaf trees and to shrubs had dropped to 20 percent in the former Zone 1 and to 4 percent in the former Zone 3. Appraisal of ponderosa pine cone production in 1936 revealed that 81 percent of the pines in Zone 1 had no cones and that outside the zone, 16 percent of the pines had no cones.[5] Sulfur dioxide concentrations 15 miles south of the smelter in Zone 1 averaged about 0.03 ppm during the summer with occasional peak concentrations in excess of 0.5 ppm for a total time of 5 to 10 hours.

Linzon [36] reported marked growth suppression and chlorotic needles on white pine at locations 25 miles and less from the Sudbury, Ontario, smelter where measurable sulfur dioxide concentrations were present from 10 percent to 20 percent of the time. Concentrations of sulfur dioxide above 0.25 ppm were present less than 1 percent of the time; the remainder of the measurable concentrations were less than 0.25 ppm. The annual average concentrations apparently ranged downward from 0.03 ppm.

Sullivan [37] reported damage to 36 percent of the home gardens in an area near a smelter in Australia where the annual average sulfur dioxide concentration was 0.009 ppm and the maximum daily average 0.155 ppm. In another area more influenced by the smelter, 89 percent of the gardens were damaged; there the annual average was 0.033 ppm and the maximum daily average 0.60 ppm. In the latter area, during brief fumigations, sulfur dioxide concentrations ranged up to 10 ppm.

Injury to at least 15 tree species, including both conifers and hardwoods, in the vicinity of petroleum refineries processing crude oil, which used 3 percent sulfur pitch residue in the refinery furnaces, was reported by Linzon.[38] Concentrations of sulfur dioxide measured in the atmosphere exceeded 0.5 ppm during 10 hours in one month in this area with momentary peaks over 2 ppm. Slight to severe acute injury was reported for all species within 1 to 2 miles from the refineries.

Reports of deteriorated vegetation in areas polluted by sulfur dioxide [23] [36] [38] make it clear that injury can occur where annual average concentrations are very low when the sources of pollution and/or the meteorological conditions are such that threshold for injury is exceeded occasionally. There is some evidence of growth suppression and particularly chronic injury, where concentrations never exceed 0.1 ppm.[29]

In Eastern Tennessee some mortality of white pine has occurred from a disease called post-emergence chronic tipburn. The disease has been noted only in industrial areas, generally within about 20 miles of plants with substantial stack emissions. The affected area in Eastern Tennessee contains several large plants, including a large coal-fired power plant, a major uranium refinement operation, a pulp mill, a ferro-alloy reduction plant, and an iron foundry. There is convincing evidence that the causal agent of the disease is atmospheric. While SO_2 was considered by some as the most likely cause of the disease, the cause was regarded by the investigators to be repeated or long continued low-level fumigations with some unidentified gas or gases produced in the affected area. Whatever the causal agent, some white pine are so sensitive to it that they have developed striking foliage symptoms at distances many miles from the pollution source.[39]

D. FACTORS AFFECTING PHYTOTOXICITY

The response of vegetation to sulfur dioxide as an air pollutant is influenced greatly by temperature, relative humidity, light intensity, soil moisture, nutrient supply, and age of plant or tissue exposed. Each of these factors is discussed below.

1. Temperature

A plant is much more resistant to sulfur dioxide at temperatures below 5° C (40° F).[21] [24] [40] [41] Setterstrom and Zimmerman [40] found that buckwheat plants were equally susceptible to injury at 65° F and 105° F. Several investigators [11] [21] [28] have reported greater resistance in winter and have related this to lower physiological activity of the plants. Physiological activity of broad-leafed plants declines sharply as the leaves become senescent and drop in fall and winter, but activity is much higher in spring and early summer. Sensitivity to sulfur dioxide injury is similarly higher in spring.[11] Weaver and Morgensen [42] found that transpiration from conifers in winter was scarcely greater than from the defoliated stems of broad-leafed trees. Apparently the conifers are essentially dormant in winter and gas exchange by the needles is very low, thus increasing resistance to toxicants.

2. Relative Humidity

Although reports of various investigators do not show good agreement, susceptibility of plants to sulfur dioxide generally tends to increase with increases in relative humidity, providing light and soil moisture are not limiting.[11, 21, 23, 40]

3. Soil Moisture

Minor variations in soil moisture during exposure have no detectable effect on susceptibility if there is sufficient moisture for normal plant growth.[40] When plants approach the wilting point, resistance increases significantly. Plants grown with an ample water supply are much more susceptible than plants grown with inadequate water.

4. Light Intensity

Plants in complete darkness are highly resistant to sulfur dioxide and the resistance decreases as the light intensity is increased up to 3,000 candles. Plants grown in shade before exposure to the toxicant are more susceptible than those grown in full sun.[13, 21, 23, 40]

5. Nutrient Supply

Alfalfa plants grown in a deficient nutrient supply were more susceptible to sulfur dioxide than plants receiving adequate nutrients.[40]

6. Age of Plant Tissue

Young plants and newer foliage are much more resistant to sulfur dioxide than older leaves. Middle-aged leaves have highest susceptibility.[40]

E. SYNERGISTIC EFFECTS OF SULFUR DIOXIDE AND OZONE

Menser and Heggestad[43] demonstrated synergistic action between sulfur dioxide and ozone. Combinations of sublethal concentrations of sulfur dioxide (0.24 ppm) and ozone (0.03 ppm) in two-hour fumigations produced injury to tobacco plants. When the exposure time was doubled (4 hours), the severity of response was approximately doubled, but there was no response when fumigation consisted of either sulfur dioxide or ozone alone. Heck[44] found moderate to severe injury on tobacco from 4-hour exposures to 0.03 ppm of ozone in combination with 0.1 ppm of sulfur dioxide. Middleton *et al.*[15] reported that the addition of small amounts of sulfur dioxide to polluted atmospheres in the Los Angeles Basin reduced the amount of injury produced in plants. He also reported that a ratio of sulfur dioxide to ozone less than 4:1 made the plants less susceptible to ozone damage; but if the ratio was 6:1 or higher, there was no evidence of interference with ozone susceptibility. Studies by Hindawi[45] substantiate the synergistic responses reported for ozone and sulfur dioxide. Heck[44] reported a synergistic reaction between nitrogen dioxide and sulfur dioxide. A 4-hour exposure to a mixture of 0.1 ppm of nitrogen dioxide and 0.1 ppm of sulfur dioxide produced moderate injury to a sensitive tobacco variety. If there is a synergistic response between ozone and sulfur dioxide or other pollutants, it offers a partial explanation for occasional inconsistencies between findings in the laboratory with single agents and findings on plant response in the natural environment.

F. EFFECTS OF SULFURIC ACID MIST ON VEGETATION

Thomas *et al.*[2] discussed experiments in which plants were exposed to sulfuric acid mists in concentrations of from 108 mg/m^3 to 2160 mg/m^3. Sulfuric acid droplets have settled on dry leaves without causing injury, but when the leaf surface was wet a spotted injury has developed. Middleton *et al.*[15] and Thomas[2] reported that this type of injury occurred in the Los Angeles area during periods of heavy air pollution accompanied by fog when the surface of the leaf may be wet. Injury may also occur in the absence of fog near combustion effluents containing sulfur oxides when the gas affluent dew point permits acid droplet formation.

The sequence of symptom development is one in which the exposed surface, usually the upper surface, shows the initial necrosis. The pH of the leaf-surface moisture may be less than three. Cellular collapse in many small spots develops progressively through the upper epidermis, mesophyll, and lower epidermis of the leaf, leaving scorched areas. No glazing or bleaching accompanies this injury, and leaf areas covered by exposed leaves show no marking. In the Los Angeles area, swiss chard and beets have shown the most typical injury (described above) of all plant species examined. Alfalfa has also developed a spotted injury pattern. Spinach is more uniformly wetted by fog and has shown a more diffuse type of injury.

G. SUMMARY

Sulfur dioxide absorbed by plants may produce two types of visible leaf injury, acute and chronic. Acute injury, which is associated with high concentrations over relatively short intervals, usually results in the injured tissues drying to an ivory color, but sometimes they darken to a reddish-brown. Brownish discoloration occurs in the tips of pine needles, which may be followed by abscission, while broad-leaved plants and grasses show necrotic blotching. Chronic injury, which results from lower concentrations over a number of days or weeks, leads to a gradual yellowing (i.e., chlorosis, in which the chlorophyll-making mechanism is impeded) or pigmentation of leaf tissue. Generally, for growth and production to be affected, visible symptoms of injury, such as acute lesions, chronic chlorosis, or excessive leaf drop, also occur.

The mechanism by which acute injury occurs apparently involves the plant's ability to transform absorbed sulfur dioxide into sulfuric acid and then into sulfates which are deposited at the tip or edges of the leaf; with a high rate of absorption, sulfite is thought to accumulate, and sulfurous acid is then formed which subsequently attacks the cells. Chronic injury, on the other hand, results from the gradual accumulation of excessive amounts of sulfate in the leaf tissue. Sulfate formed in the leaf from sulfur dioxide absorbed from the atmosphere is additive to sulfate absorbed through the roots. Although levels of sulfate in some leaf tissue five to ten times above normal have been noted with no detectable symptoms of injury, very high levels cause chronic symptoms and stimulate leaf drop.

The amount of acute injury caused by sulfur dioxide depends on the absorption rate of the gas, which is a function of the concentration. Thus a given amount of gas obsorbed in a short period (at a high concentration) will cause more leaf destruction than if the same amount of gas were absorbed over a longer period (at a lower concentration). Mathematical expressions have been worked out which, for some plant species, may be used to relate concentration, time of exposure, and amount of damage. Different varieties of plants vary widely in their susceptibility to sulfur dioxide injury. The threshold response of alfalfa to acute injury is 1.25 ppm over one hour, whereas privet requires 15 times this concentration for the same amount of injury to develop. Some species of trees and shrubs have shown injury at exposures of 0.5 ppm for seven hours, while injury has been produced in other species at 3-hour sulfur dioxide exposures of 0.54 ppm and, in still others, at 8-hour exposures of 0.3 ppm. From such studies, it appears that acute symptoms will not occur if the maximum concentration for the year does not exceed 0.3 ppm. From the data on the CAMP cities (Chapter 3), an 8-hour maximum concentration of 0.3 ppm would correspond to a mean annual concentration of between 0.03 ppm and 0.05 ppm.

Chronic symptoms and excessive leaf drop may be produced by long-term exposures to lower concentrations and have been reported in locations where the mean annual concentration is below about 0.03 ppm. Because chronic injury results from the slow buildup of sulfate in the tissue, leaf sulfate analyses may be a useful index for evaluating chronic injury.

The suppression of growth and yield usually is accompanied by visible symptoms of injury. A straight-line relationship, for example, has been found between the yield of alfalfa and the total area destroyed by acute symptoms or the area covered by the chlorotic symptoms. However, it has been suggested that, in some cases, sulfur dioxide might suppress growth and yield without causing visible injury. One investigator reported that yields of rye grass grown in unfiltered air were significantly lower than similar plants grown in filtered air, with no visible symptoms in the plants. Sulfur dioxide levels in the unfiltered air ranged from 0.01 ppm to 0.06 ppm with exposure periods ranging from 46 to 81 days, although it is not known whether other gaseous pollutants were present.

Sensitivity of plant materials is affected significantly by such environmental conditions as temperature, relative humidity, soil moisture, light intensity, nutrient level, and by sulfate content of the soil and irrigation water. At locations in the vicinity of point sources of sulfur dioxide, high concentrations occur with greater frequency and damage to plants is more likely to occur. Plant damage has been noted as much as 52 miles downwind from a smelting operation which emitted large quantities of sulfur dioxide. Slight-to-severe injury was reported to at least 15 tree species, including both conifers and hardwoods, in the vicinity of petroleum refineries where concentrations exceeded 0.5 ppm during 10 hours in one month with momentary peaks as high as 2 ppm.

Sulfur dioxide concentrations from 0.05 to 0.25 ppm will react synergistically with either ozone or nitrogen dioxide in short term exposures (e.g., 4 hours) to produce moderate-to-severe injury on certain sensitive plants.

Sulfuric acid mist, which may occur in polluted fogs and mists, also damages leaves. The acid droplets cause a spotted injury to leaves which are wet due to fog conditions. Such injury may occur at concentrations of 0.1 mg/m^3.

H. REFERENCES

1. Thomas, M. D. "Gas Damage to Plants." Ann. Rev. Plant Physiol., Vol. 2, pp. 293–322, 1951.
2. Thomas, M. D., Hendricks, R. H., and Hill, G. R. "Some Impurities in the Air and Their Effects on Plants." In: Air Pollution, L. C. McCabe (ed.), McGraw-Hill, New York, 1952, pp. 41–47.
3. Thomas, M. D. and Hendricks, R. H. "Effect of Air Pollutants on Plants." In: Air Pollution Handbook, McGraw-Hill, New York, 1956.
4. Thomas, M. D. "Effects of Air Pollution on Plants." In: Air Pollution, W. H. O. Monograph Series 46, Columbia Univ. Press, New York, 1961, pp. 233–275.
5. Sheffer, T. C. and Hedgcock, G. G. "Injury to Northwestern Forest Trees by Sulfur Dioxide from Smelters." U.S. Dept. Agri., Forest Service, Tech. Bull. 1117, 1955.
6. Brandt, C. S. and Heck, W. W. "Effects of Air Pollutants on Vegetation." In: Air Pollution, 2nd edition, Vol. I, A. C. Stern (ed.), Academic Press, New York–London, 1968, pp. 401–443.
7. Katz, M. and McCallum, A. W. "The Effect of Sulfur Dioxide on Conifers." In: Air Pollution, Chapt. 8, L. C. McCabe (ed.), 1952, pp. 84–96.
8. Daines, R. H. "Sulfur Dioxide and Plant Response." Preprint. (Presented at Symposium on Air Quality Criteria, New York, June 5, 1968.)
9. Wood, F. A. J. Occup. Med., Vol. 10, pp. 92–102, 1962. (Discussion at Symposium on Air Quality Criteria, New York, June 4–5, 1968.)
10. Altman, P. L. and Dittner, D. S. (eds.) "Environmental Biology (Biological Handbooks)." Federation of American Societies for Experimental Biology, Bethesda, Maryland, 1966, p. 315.
11. Katz, M. "Sulfur Dioxide in the Atmosphere and its Relation to Plant Life." Ind. Eng. Chem., Vol. 41, pp. 2450–2465, 1949.
12. Holmes, J. A., Franklin, E. C., and Gould, R. A. "Selby Report." U.S. Bureau of Mines, Bulletin 98, 1915.
13. Solberg, R. A. and Adams, D. F. "Histological Response of Some Plant Leaves to Hydrogen Fluoride and Sulfur Dioxide." Amer. J. Bot., Vol. 43, pp. 755–760, 1956.
14. Thomas, M. D., Hendricks, R. H., and Hill, G. R. "Sulfur Metabolism of Plants: Effect of Sulfur Dioxide on Vegetation." Ind. Eng. Chem., *42*(11):2231–2235, 1950.
15. Middleton, J. T., Darley, E. F., and Brewer, R. F. "Damage to Vegetation from Polluted Atmospheres." J. Air Pollution Control Assoc., Vol. 8, pp. 9–15, 1958.
16. Thomas, M. D. and Hill, G. R. "Relation of Sulfur Dioxide in the Atmosphere to Photosynthesis and Respiration of Alfalfa." Plant Physiol., Vol. 12, pp. 309–383, 1937.
17. Brennan, L. and Daines, R. H. "Investigation of SO_2 Effects on Rubber Trees as a Means of Forestalling Injury to Malayan Plantations from Refinery Emissions." J. Air Pollution Control Assoc., Vol. 14, pp. 229–233, 1964.
18. Massey, L. M. "Similarities Between Disease Symptoms and Chemically Induced Injury to Plants." In: Air Pollution, Chapt. 3, L. C. McCabe, (ed.), McGraw-Hill, New York, 1952, pp. 48–58.
19. "Plants Absorb Sulfur Dioxide, Release Hydrogen Sulfide." Sulfur Institute J., *4*(2):17, 1968.
20. Stoklasa, J. "Die Beschadigung der Vegetation durch Rauchgase und Fabriksexhalationen." Urban und Schwartzenberg, Berlin, 1923.

21. Setterstrom, C. "Effects of Sulfur Dioxide on Plants and Animals." Ind. Eng. Chem., Vol. 32, pp. 473–479, 1940.

22. Swain, R. E. and Johnson, A. B. "Effect of Sulfur Dioxide on Wheat Development; Action at Low Concentration." Ind. Eng. Chem., *28*(1): 42–47, 1936.

23. Wells, A. E. "Results of Recent Investigations of the Smelter Smoke Problem." Ind. Eng. Chem., Vol. 9, pp. 640–646, 1917.

24. Zimmerman, P. W. "Chemicals Involved in Air Pollution and their Effects upon Vegetation." Professional Papers, Boyce Thompson Institute, New York *2*(14):124–145, 1955.

25. Thomas, M. D. and Hill, G. R. "Adsorption of Sulfur Dioxide by Alfalfa and its Relation to Leaf Injury." Plant Physiol., Vol. 10, pp. 291–307, 1935.

26. "Effects of Sulfur Dioxide on Vegetation." National Research Council of Canada, Ottawa, Pub. 815, 1939, p. 447.

27. Whitby, G. S. "The Effects of Sulfur Dioxide on Vegetation." Chem. Ind., Vol. 58, p. 99, 1939.

28. Matushima, J. and Horada, M. "Sulfur Dioxide Gas Injury to Fruit Trees. V. Absorption of Sulfur Dioxide by Citrus Trees and its Relation to Leaf Fall and Mineral Contents of Leaves." J. Soc. Hort. Sci. (Tokyo), *35*(3):241–246, 1966.

29. Bleasdale, J. K. A. "Atmospheric Pollution and Plant Growth." Nature, Vol. 169, p. 376, 1952.

30. Haselhoff, E. and Lindau, G. "Die Beschadigung der Vegetation durch Rauch." Verlagsbuchhandlung Gebrueder Borntraeger, Leipzig, 1903, p. 412.

31. Dorries, W. "Uber die Brauchbarkeit der spectroscopischen Phaophytinprobe in der Rauchschaden-Diagnostik." Z. Pflanzenkrankheiten u. Gallenkunde, Vol. 42, pp. 257–273, 1932.

32. Noack, K. "Damage to Vegetation from Gases in Smoke." Z. Angew. Chem., Vol. 42, pp. 123–126, 1929.

33. O'Gara, P. J. Ind. Eng. Chem., Vol. 14, p. 744, 1922.

34. Guderian, R., von Haut, H., and Stratman, H. Z. Pflanzenkrankh. Pflanzenschutz, Vol. 67, p. 257, 1960. (Quoted in ref. 6, p. 421.)

35. Spierings, F. "Method for Determining the Susceptibility of Trees to Air Pollution by Artificial Fumigation." Atmos. Environ., Vol. 1, pp. 205–210, 1967.

36. Linzon, S. N. "The Influence of Smelter Fumes on the Growth of White Pine in the Sudbury Region." Ontario Dept. of Lands and Forest, Ontario Dept. of Mines, Toronto, 1958, pp. 1–45.

37. Sullivan, J. L. "The Nature and Extent of Pollution by Metallurgical Industries in Port Kembla." In: Air Pollution by Metallurgical Industries, Dept. of Public Health, Div. of Occupational Health, Sydney, Australia, Vol. 1, pp. 1–59, 1962.

38. Linzon, S. N. "Sulfur Dioxide Injury to Trees in the Vicinity of Petroleum Refineries." Forest Chronicle, Vol. 41, pp. 245–250, 1965.

39. Berry, C. R. and Hepting, G. H. "Injury to Eastern White Pine by Unidentified Atmospheric Contaminants." Forest Sci., Vol. 10, pp. 2–13, 1964.

40. Setterstrom, C. and Zimmerman, P. W. "Factors Influencing Susceptibility of Plants to Sulfur Dioxide Injury." Boyce Thompson Institute, New York, Vol. 10, pp. 155–181, 1939.

41. Swain, R. E. "Atmospheric Pollution by Industrial Wastes." Ind. Eng. Chem., Vol. 15, pp. 296–301, 1923.

42. Weaver, J. E. and Morgenson, A. "Relative Transpiration of Coniferous and Broad-Leafed Trees in Autumn and Winter." Bot. Gaz., Vol. 68, pp. 393–424, 1919.

43. Menser, H. A. and Heggestad, H. E. "Ozone and Sulfur Dioxide Synergism: Injury to Tobacco Plants." Science, Vol. 153, pp. 424–425, 1966.

44. Heck, W. W. "Discussion of O. C. Taylor's Paper: Effects of Oxidant Air Pollutants." J. Occup. Med., Vol. 10, pp. 497–499, 1968.

45. Hindawi, I. J. "The Effects of Sulfur Dioxide on Vegetation Grown in the Washington, D.C. Metropolitan Area." Preprint. (Presented at the Washington, D.C. Area Air Pollution Abatement Activity Conference, Dec. 13, 1967.)

BLANK PAGE

Chapter 6

TOXICOLOGICAL EFFECTS OF SULFUR OXIDES ON ANIMALS

Table of Contents

Page

A. INTRODUCTION 73

B. EVALUATION BY MORTALITY AND PATHOLOGY 73
  1. Sulfur Dioxide Exposure 73
  2. Sulfuric Acid Aerosol 75

C. EVALUATION BY PULMONARY FUNCTION 76
  1. Sulfur Dioxide Exposure 76
  2. Mechanism of Response 79
  3. Sulfuric Acid and Particulate Sulfate Exposures 80

D. EFFECTS OF SULFUR DIOXIDE ON CILIARY ACTION 81

E. LIFETIME EXPOSURE OF ANIMALS TO SULFUR DIOXIDE 82

F. ABSORPTION AND DISTRIBUTION OF SULFUR DIOXIDE 82

G. MISCELLANEOUS BIOCHEMICAL EFFECTS OF SULFUR DIOXIDE 84

H. SUMMARY 84

I. REFERENCES 85

Chapter 6

TOXICOLOGICAL EFFECTS OF SULFUR OXIDES ON ANIMALS

A. INTRODUCTION

The toxicology of oxides of sulfur is reviewed in Chapters 6 to 8 of this document. Unfortunately, published data generally refer to concentrations far in excess of those likely to be found in polluted atmospheres, and therefore have little relevance to criteria for atmospheric pollutants. Even where the animal studies are carried out at more realistic levels, it is certain that the health hazard associated with community air pollution can only be evaluated directly in epidemiologic terms (chapter 9). Nevertheless, toxicological experiments do indicate the kinds of physiological and pathological response of which animals and man are capable. Studies have been chosen for review in Chapters 6 to 8 on the basis of their possible relevance to air pollution problems, and the chapters are not comprehensive treatments of sulfur oxide toxicology.

While unreliable conversions and extrapolations are necessary in assessing health effects on man from toxicological experiments on animals, such experiments do, however, permit the use of higher concentrations, longer exposure times, larger numbers of subjects, surgical procedures, postmortem examinations, and other liberties not open to the experimenter with human subjects. Since man is an animal, the results of such animal experiments should have some relation to man, even though it is only qualitative. Therefore, this chapter deals with the toxicology of the oxides of sulfur in animals.

Methods of evaluation include challenging animals with the pollutant or pollutant combinations and examining mortality and changes in lung function that precede irreversible pathology. Absorption, distribution, and retention of sulfur oxides have also been examined, as have biochemical change and ciliary response in the organism. The effects of oxides of sulfur on animals have been reviewed by Setterstrom [1] and by Negherbon.[2]

B. EVALUATION BY MORTALITY AND PATHOLOGY

1. Sulfur Dioxide Exposure

The response of various species including guinea pigs, mice, grasshoppers, and cockroaches to sulfur dioxide [1 3 4 5] has been studied. Concentrations from 10 ppm to 1,000 ppm (~30 mg/m^3 to 30,000 mg/m^3) were monitored and duplicate chambers were used for control animals.[6] Exposures were continuous up to the time of death of 50 percent of the animals or until no progressive symptoms appeared after a considerable time. At concentrations of 25 ppm (~72 mg/m^3) no mortality was produced by treatment up to about 45 days. At concentrations of 150 ppm (430 mg/m^3) and below, mice were more resistant than guinea pigs. For example, it required 847 hours to kill 50 percent of the mice at 150 ppm, whereas 50 percent of the guinea pigs exposed to 130 ppm (~370 mg/m^3) died in 154 hours. At concentrations of 300 ppm (~860 mg/m^3) to 1,000 ppm mice were less resistant than guinea pigs. At levels of around 1,000 ppm, 50 percent of the mice died in about 4 hours whereas about 20 hours were needed to kill guinea pigs. The susceptibility of the two insect species approximated that of the mice. It is thus apparent that extrapolations from high to lower concentrations of such parameters as species sensitivity are frequently not reliable.

No significant mortality or signs of distress were noted among healthy animals exposed to concentrations of 33 ppm (94 mg/m^3) or below. Longer exposure was needed to cause mortality when the exposures were intermittent. Exercise increased susceptibility of guinea pigs exposed to 1,000 ppm but had no effect at lower concentrations.

Symptoms produced by the higher concentrations included coughing, moderate dyspnea, rhinitis, lachrymation, conjuctivitis, abdominal distension, lethargy, weakness, and paralysis of the hind quarters. Pathologic changes included slight to moderate visceral congestion, slight to moderate pulmonary edema, and distension of the gall bladder and stomach. Higher concentrations produced hemorrhages of lungs and stomach and acute dilation of the right heart.

The mean survival time of mice, guinea pigs, and rats was measured as a means of evaluating the effect of pretreatment with histamine, or of adrenalectomy, on the toxicity of sulfur dioxide.[7] Concentrations in the range of 600 ppm to 5,000 ppm (1700 mg/m^3 to 14,000 mg/m^3) were used, presumably because it was the aim to kill normal animals within a convenient time interval. For normal animals the susceptibility was highest in mice, intermediate in guinea pigs and least in rats. At the concentrations used in these experiments the relative susceptibility of mice and guinea pigs is in agreement with the earlier data of Weeden *et al.*[2] mentioned above. Histamine given by intraperitoneal injection at levels of 200 mg/kg of animal weight 10 minutes before exposure significantly decreased the survival time of rats and mice. Adrenalectomy in rats also reduced survival time.

The extent of changes in lung pathology and the amount of fluid in the alveoli were primarily dependent upon the exposure time. In normal animals, pulmonary edema and areas of consolidation were usually observed, while animals whose survival time had been reduced by pretreatment, exhibited bronchial obstruction. Two mechanisms of death were observed in guinea pigs. A portion of each test group succumbed rapidly when introduced into the sulfur dioxide atmosphere. Occlusion of the bronchioles and venous congestion with little or no fluid in the alveoli were the pathological changes observed in these animals. Those animals which survived 2 to 4 hours showed fluid in the alveoli, distended bronchioles and partially desquamated mucosal membranes.

Goldring *et al.*[9] reported that 10,000 ppm (29.000 mg/m^3) produced 100 percent mortality of Syrian hamsters within 8 to 10 minutes. Exposures to 3,250 ppm (9,300 mg/m^3) for periods up to 1½ hours produced 50 percent mortality within 24 hours. Exposures of 650 ppm (1,850 mg/m^3) for 75 days did not produce significant histopathological changes in the pulmonary tree of hamsters. Sodium chloride aerosol was also present in all exposures. (See Chapter 8 and *Air Quality Criteria for Particulate Matter*, a companion document, for a discussion of synergistic effects.)

A limited number of swine was exposed to 5, 10, 20, and 40 ppm (about 14, 29, 57, and 114 mg/m^3) sulfur dioxide for 8 hours.[10] Symptoms at 5 ppm were limited to slight eye irritation and excess salivation. At higher concentrations there was excess salivation, excess nasal secretion and shallower, more rapid respiration during the first 4 hours, after which the symptoms abated. Twenty-four hours after exposure the lungs showed hemorrhage and emphysema. The changes were less marked in animals killed a week after exposure. Two animals exposed to 40 ppm and one animal exposed to 20 ppm, which were killed 160 days after exposure, showed some areas of fibrosis, and the lungs were a darker color and less well inflated than in normal animals.

Reid[11] attempted to produce hypertrophy of the goblet cells and mucous glands of rats which would simulate chronic bronchitis. Initially, animals were exposed 5 hours a day, 5 days a week, to about 40 ppm sulfur dioxide. Since no great change was found in the lungs after 3 months, the dose was increased to 300 ppm to 400 ppm (about 900 mg/m^3 to 1,100 mg/m^3), in order to produce the desired changes. There followed an increase in the number of mucus-secreting cells both in the main bronchi, where goblet cells are normally frequent, and in the fine peripheral airways from which they are normally absent. Excessive mucus was seen in the bronchial lumen, probably reflecting an increased number of mucus-secreting cells as well as greater secretory activity, associated with reduced ciliary activity leading to retention. The changes apparently were not mediated through infection, as pathogens were not more frequently found in the bronchi and lungs of animals exposed to the irritant than in the controls. Although infection may act as an irritant, these experiments indicate that it is by no means an essential factor in the development of excessive mucous cells characteristic of chronic bronchitis.

In one experiment, the reaction was great in all but 2 animals, whereas in another experiment only half the animals responded. At the time of the diminished responsiveness, however, the supplier had made an attempt to breed a strain of animals less susceptible to bronchiectasis. It is likely that there is considerable difference in response between strains.

The excess of goblet cells persisted for at least 3 months after exposure to the irritant had ceased, even in the absence of infection. Most of the goblet cells were distended with secretion, suggesting that they discharged less mucus than during the exposure. The cessation of irritant exposure seemed to arrest the increase in cells.

Dalhamn[12] studied the morphology of tracheal mucosa in rats following exposures for periods of 18 to 67 days to about 10 ppm (about 30 mg/m^3) sulfur dioxide. Exposures were 6 hours a day for 5 days a week, and 3 hours on Saturday; there was no exposure on Sunday.

Severe morphologic changes of the epithelium and lamina propria were observed. The epithelial surface was irregular with deep crypts, probably produced by cell proliferation. Since the cilia lining these crypts could not contribute to transport of mucus, such changes were a probable cause of observed retarded mucus flow. The changes were present at 18 days as well as at 67 days, and they had not regressed 4 weeks after exposure in six animals examined at that time. Support is thus lent to Reid's finding[11] of slow reversibility. There was no alteration of the ciliary ultrastructure although the surrounding tissues including ciliary cells showed severe changes. There was more mucus in the trachea of exposed animals. In healthy rats the mucus blanket was about 5-μ thick and the secretion contained only a few cellular elements. After sulfur dioxide exposure the mucus blanket was 20-μ to 25-μ thick and the secretion appeared more compact and contained numerous elements such as shed cells and white blood cells. Since the trachea is the main site of absorption of sulfur dioxide these changes are not surprising at 10 ppm.

2. Sulfuric Acid Aerosol

Treon *et al.*[13] exposed rabbits, rats, mice, and guinea pigs to sulfuric acid mist of which 95 percent was below 2 μ. Only a few animals were used in the exposures, and the concentrations were at levels of 87 to 1,600 mg/m^3; nevertheless, a clear-cut species difference emerged. The order of increasing sensitivity was: rabbits, rats, mice, and guinea pigs. Mathur and Olmstread[14] also found that mice were much more resistant than guinea pigs.

Amdur *et al.*[15] found the 8-hour LC_{50} of sulfuric acid (MMD 1-μ) to be 18 mg/m^3 for one- to two-month-old guinea pigs and 50 mg/m^3 for 18-month-old animals. Pattle *et al.*[16] determined the 8-hour LC_{50} for 200 g to 250 g guinea pigs exposed to sulfuric acid of two particle sizes, MMD 2.7-μ and MMD 0.8-μ. The LC_{50} was 27 mg/m^3 for the large particles and 60 mg/m^3 for the small particles. At 0° C with 0.8-μ particles the LC_{50} was 47 mg/m^3, a value significantly different from that at room temperature. The guinea pig is a tropical animal and the difference in effect is considered a direct action of cold on the animals. Ammonium carbonate sufficient to provide an excess of ammonia in the chamber gave protection from levels of sulfuric acid which, in the absence of ammonia, would have caused 50 percent mortality, emphasizing that the toxicity is related to acidity.

The pathological findings were similar in the two investigations. Animals dying after short exposure (less than 2 hours) show grossly distended and emphysematous lungs with no other serious lesions.[16] The cause of death in these animals appeared to be asphyxia caused by bronchoconstriction and laryngeal spasm. Animals dying after longer exposures showed gross pathological lesions including capillary engorgement and hemorrhage.[15, 16] It is suggested that these observations were mainly sequelae of the combined effects of anoxia and increased intrathoracic pressure caused by bronchoconstriction and laryngeal spasm.[16] That animals succumbing quickly show a pathology different from those surviving longer exposures is similar to the findings of Leong *et al.*[7] for guinea pigs exposed to massive concentrations of sulfur dioxide. The animals that survived the exposures showed spotty areas of old hemorrhage and some areas of consolidation, especially around the hilar regions. Such damage to the animals surviving an exposure lethal to others is repaired very slowly.

Amdur *et al.*[15] next extended exposure times to 72 hours and found that the increased exposure did not produce mortality at 8 mg/m^3, nor did it, at higher concentrations, increase the mortality beyond that which was observed in 8-hour exposures. In the 8-hour exposures, most of the animals that died did so within the first 4 hours. Thickening of the alveolar walls and areas of consolidation were much greater in animals exposed for 72 hours to 8 mg/m^3 than in animals exposed for 8 hours. It was postulated that sulfuric acid has two distinct toxic actions:

1. It promotes laryngeal and bronchospasm as the cause of death,
2. It can also cause parenchymal lung damage; this action is dependent on total dosage.

As Pattle *et al.*[16] have observed, the damage is probably a direct irritant effect of the acid mist deposited on the lung tissue and may be the mechanism by which the mist causes death in those animals more resistant than the guinea pig to brochospasm.

Longer exposure times ranging from 18 to 40 days were used by Thomas *et al.*[17] with particle sizes of 0.6μ, 0.9μ, and 4μ and with concentrations up to 4 mg/m^3. The ages of the animals were not given, but old animals were less sensitive to the pathological effects of the mist than the young animals. This is in agreement with acute toxicity based on LC_{50}.[15] Of the three sizes used, 0.9μ particles produced greater change than either the larger or the smaller ones. Lesions of the upper respiratory tract consisting of a slight edema of the larynx and trachea and a decrease in mucus in the major bronchi, were observed with the 4-μ particles. In the young animals exposed to 0.9-μ acid, the lungs were slightly edematous, and a rare capillary hemorrhage was noted. In minor, but not in major, bronchi there was some increase in desquamated epithelial cells, and the epithelium appeared intact; both major and minor bronchi showed a reduced amount of mucus and less spasm than controls. The spasm in the controls was presumably a result of sacrificing the animals by chloroform inhalation. On the whole, the observed pulmonary pathology was slight, and it was concluded that the guinea pig can tolerate levels of about 2 mg/m^3 for more than 3 months of continuous exposure with only minor pathological effects.

Bushtueva[18, 19] reported that guinea pigs exposed to 2 mg/m^3 of sulfuric acid mist of unspecified particle size for 5 days developed edema and thickening of the alveolar walls. One- to 2-week exposures to 1 mg/m^3 produced a slight catarrhal condition of the mucous membranes of the trachea and bronchi and slightly defined, but widely distributed interstitial proliferative processes accompanied by round lymphoid cell infiltration surrounding the blood vessels and bronchi. These latter changes seemed progressive as they were more highly developed 2 to 3 months after exposure. A concentration of 0.5 mg/m^3 produced only a slight lung irritation.

C. EVALUATION BY PULMONARY FUNCTION

1. Sulfur Dioxide Exposure

In a series of papers, Amdur and her coworkers have reported on the respiratory response of guinea pigs to various irritant air pollutants, among them sulfur dioxide.

The methods employed in all these studies are documented in the original paper dealing with sulfur dioxide [20] and in a paper dealing with physiological methods of measurement.[21] The method has the advantage that the animal is unanesthetized and breathing spontaneously. The information obtained in these experiments is, however, limited in comparison with studies of irritant action using anesthetized larger species. The method is suitable for routine daily use by a carefully trained technician, and data on numbers of animals exposed only once to a specific irritant can readily be obtained. This eliminates the need for re-exposure following "recovery" which is necessary in complicated physiological experiments.

Amdur and her co-workers used a standard exposure time of 1 hour; thus the applicability to air pollution criteria would be in connection with the possible effects of short-term peak values. Although the animals were unanesthetized during the control and exposure periods, an intrapleural catheter was used, and this was put in place with the animals under light ether anesthesia. Mead [22] used another method which measured flow resistance without the need for an intrapleural catheter to measure the resistance of guinea pigs before and after intubation, and found that the procedure had caused an increase. It was not determined whether this increase was caused by the presence of the catheter or by the recent exposure to ether.

The method yields information on pulmonary flow-resistance and compliance (a measure of the elastic behavior of the lungs),[20, 23] as well as tidal volume, respiratory frequency, and minute volume. One-hour exposures produced statistically significant increases in resistance ranging from about 10 percent at 0.16 ppm (460 $\mu g/m^3$) to about 265 percent at 835 ppm (2,390 mg/m^3).[24] For most normal human subjects, an increase in resistance of 300 percent or less would have no marked physiological consequences.

After exposure was terminated, resistance decreased and at concentrations up to 100 ppm (~290 mg/m^3) it returned to pre-exposure levels in the course of an hour. Resistance was back to normal 2 hours after exposure to 300 ppm (860 mg/m^3).[25] A decrease in respiratory frequency was observed, and it became statistically significant at concentrations above 25 ppm.

The pattern of response produced by sulfur dioxide is typical [23] of the response of the guinea pig to a group of respiratory irritants which includes acetic acid, formaldehyde, and droplets of sulfuric acid smaller than 1μ. At levels of 2 ppm (~6 mg/m^3 to 14 mg/m^3), the other irritants all evoked a greater response than did sulfur dioxide. For this group of irritants, increased flow resistance is the most sensitive indicator of response.

Pulmonary edema is a relatively minor factor in the pathology of sulfur dioxide and similar irritants (although it is important for ozone and oxides of nitrogen).[26] The patterns of pathological response may relate to the ability of the irritant to penetrate to peripheral areas of the lung. Penetration is an important factor, but is not the only explanation, since irritant aerosols, of a size expected to penetrate readily, produced the pattern typified by sulfur dioxide and aldehydes.[27]

Amdur and her co-workers also measured the response of guinea pigs breathing irritants through a tracheal cannula.[20, 23, 24] The response to a given concentration of sulfur dioxide was greater when the gas reached the lungs through a tracheal cannula at concentrations of 2 ppm (~6 mg/m^3) and above. At concentrations near 0.4 ppm to 0.5 ppm (1.1 mg/m^3 to 1.4 mg/m^3) the response was not altered by the tracheal cannula. This supports the data of Strandburg [28] indicating that low concentrations of sulfur dioxide are not efficiently removed by the upper respiratory tract.

Exposures of guinea pigs breathing normally and via a tracheal cannula to sulfur dioxide at 5 ppm to 10 ppm (about 14 mg/m^3 to 29 mg/m^3) are reported by Davis *et al.*[29] Exposures of normal animals produced increases in resistance and tidal volume and decreases in respiratory rate and minute volume. Insufficient data are given to evaluate the specific effects of the irritant. There were no changes in respiratory function in animals exposed via tracheal cannula. Such animals did, however, respond with a resistance increase to inhalation of histamine aerosol. The authors conclude that sulfur dioxide is without effect on the lung and the entire response seen in normal animals is due to an increase in resistance of the upper airways.

This interpretation may be questioned since the pulmonary function tracings show evidence of technical errors where flow and volume appear completely out of phase with the pressure tracing. The results are not in agreement with those of Amdur and Mead [20] and of Amdur [23] obtained on guinea pigs, nor with the results discussed below for other species.

Balchum *et al.*[30-33] exposed anesthetized dogs to sulfur dioxide by nose and mouth breathing and by means of a tracheal cannula. Concentrations ranged from around 1 ppm to around 140 ppm (about 3 mg/m^3 to 400 mg/m^3), and exposure periods were 20 minutes to 40 minutes. The dogs were not breathing spontaneously, but were ventilated at constant stroke volume following a dose of intravenous nembutal sufficient to stop spontaneous respiration. This represents an unusually deep level of anesthesia. A decrease in compliance and an increase in resistance was found at all concentrations used. These alterations were also produced when only an isolated segment of trachea was exposed to sulfur dioxide.[30] The resistance change was greatest in animals breathing via a tracheal cannula, intermediate in those nose-breathing, and least in those dogs in which only a tracheal segment was exposed. The compliance changes, on the other hand, were of the same order of magnitude in all cases. The compliance changes were possibly caused by some extraneous influence and not by sulfur dioxide, since spontaneous compliance decreases are a common phenomenon in dogs lying on their backs during prolonged physiological experiments.[31]

Salem and Aviado [35] ventilated dogs by pump through a tracheal cannula and measured the "dynamic" pressure-volume char-
200 ppm. to 850 ppm (570 mg/m^3 to 2400 mg/m^3) sulfur dioxide for periods of 1 to 4 minutes. The changes that ensued were
acteristics of the lungs during exposures of consistent with bronchoconstriction preceded and followed by bronchodilation. These changes were interpreted as protective measures leading to a reduction in the amount of sulfur dioxide absorbed via the pulmonary circulation. They also observed pulmonary vasoconstriction, increase in pulmonary arterial blood pressure, a depression of myocardial force of contraction accompanied by brachycardia, and systemic shock.

Yokoyama and Ishikawa [36] measured the effect of sulfur dioxide on the mechanical behavior of the lungs of dogs exposed to 50 ppm to 350 ppm (140 mg/m^3 to 1,000 mg/m^3) via a tracheal cannula. There was consistent increase, occasionally as much as tenfold, in flow resistance. There were no consistent changes in pulmonary compliance.

Frank and Speizer [37] compared the functional response of several levels of the respiratory system to inhaled sulfur dioxide. Dogs were lightly anesthetized and exposed by nose, by tracheal cannula, and by an isolated segment of trachea. Concentrations ranged from 7 ppm to 230 ppm (20 mg/m^3 to 660 mg/m^3), and routine exposures were for 15 minutes. Intervals of at least 20 minutes were allowed between exposures, and no animal was exposed more than 4 times. In eight animals exposed by nose the nasal resistance was measured. At levels of 7 ppm to 61 ppm (175 mg/m^3), the nasal resistance increased in a manner roughly proportional to the concentration, and at concentrations above 25 ppm (about 70 mg/m^3) it became progressively greater with duration of exposure. One animal showed a reduction in nasal resistance in the first 2 exposures and an increase in the second 2 exposures. During recovery, which lasted 15 to 40 minutes, the values reverted partially or completely to control values. The authors speculate that these changes probably reflect mucosal swelling, increased secretion of mucus or both. The resistance of the larynx appeared to rise and fall about an equal number of times during exposure and recovery. A significant change in one direction or the other occurred in about 45 percent of the observations. During nose-breathing the resistance of the lungs rose and fell about an equal number of times and the direction of change was not related to concentration of gas nor to duration of exposure. About one-third of these changes were considered statistically significant. Significant changes in either direction occurred more frequently at higher concentrations. During recovery the lung resistance tended to increase above control levels. Compliance was only slightly lower during exposure. Minute ventilation fell owing to slight reduction in both tidal volume and respiratory frequency.

When exposure was by tracheal cannula, pulmonary flow resistance rose rapidly, reaching a peak within several minutes, and then decreased. Limiting the exposure to the larynx and upper trachea produced a much less pronounced increase in lung resistance.

2. Mechanism of Response

Basic information on the mechanism of response to irritants is important in the complex task of assessing the irritant potential of air pollution. This is the realm of the physiologist, who may, for example, experiment with the effects of an irritant material on nerve fibers, or with the specific blocking effect of drugs. Exposures are usually brief, sometimes only a single breath, and quite frequently the concentration is not specified, but can be assumed to be very high.

Widdicombe[38] studied, in cats, the cough reflex elicited by mechanical stimulation, powdered talc or starch, and sulfur dioxide. He found that sulfur dioxide elicited the cough reflex when given through an endobronchial catheter so that the gas came in contact only with the lungs and smaller bronchi, and not with the trachea and extrapulmonary bronchi. The cough reflex produced in this way was always stronger than when the gas was applied to the trachea and main bronchi alone. After several inhalations of sulfur dioxide the cats became completely refractory to the gas but gave normal responses to mechanical stimulation of the trachea. This result suggested that the receptors sensitive to mechanical stimuli were not being stimulated by sulfur dioxide. Further evidence that the two receptors were distinct was provided by the fact that procaine solution sprayed into the trachea blocked the mechanical cough reflex but did not affect the response to sulfur dioxide.

Widdicombe[39] also made a systematic examination of different nerve endings in the tracheobronchial tree. One hundred single vagal fibers, which were excited by inflation of the lungs, were dissected, and the response was studied under various conditions. One group of these fibers was first sensitized, then inhibited, by sulfur dioxide. This particular group of fibers was distributed throughout the lower trachea and main bronchi. The responses of these fibers were not inhibited by procaine; this results parallels the observation that the cough reflex elicited by sulfur dioxide is not inhibited by procaine.[24] Another group of fibers was sensitive to mechanical stimuli but insensitive to sulfur dioxide; this sensitivity parallels the finding[24] that the mechanical cough reflex was still present in animals rendered refractory to sulfur dioxide. Vagal temperatures of 7° C to 10° C blocked the fibers sensitive to sulfur dioxide. As with mechanical stimuli, sulfur dioxide acts via the sympathetic nervous system as well as the vagus nerves, and the group of fibers sensitive to sulfur dioxide has been shown to connect with the sympathetic trunk.

The mechanism of bronchoconstriction produced by sulfur dioxide in cats was also examined.[40] The animals were anesthetized, the respiratory muscles were paralyzed with gallamine triethiodide, and the animals were ventilated with a pump via a tracheal cannula. Sulfur dioxide was delivered either to the lungs or to the upper airways. Total pulmonary resistance was measured. Sulfur dioxide delivered to the lower airways and lungs during a single inflation cycle produced an increase in resistance which began during the first breath after exposure and returned to control levels within 1 minute. Exposing only the upper airways also produced a resistance increase. During cooling of the cervical vagosympathetic nerves the response was abolished whether the sulfur dioxide was delivered to the lung or to the upper airways. After rewarming of the nerves, the response was re-established. The response was also abolished by the intravenous injection of atropine. The rapidity of the response and its reversal suggests that changes in smooth muscle tone are the cause of the bronchoconstriction. This response depends upon intact motor parasympathetic pathways. Similar results were reported by Widdecombe *et al.*[39, 41] for bronchoconstriction caused by inhalation of fine charcoal dust.

3. Sulfuric Acid and Particulate Sulfate Exposures

The respiratory response of guinea pigs to sulfuric acid mist was studied by Amdur.[42] Concentrations ranged from 2 mg/m^3 to 40 mg/m^3. Three particle sizes were used, 0.8μ, 2.5μ, and 7μ MMD. The $7\text{-}\mu$ particles even at a concentration of 30 mg/m^3 caused only a slight increase in resistance and no other detectable alterations. Particles of this size would not penetrate to any extent beyond the nasal passages. The $0.8\text{-}\mu$ particles produced an increase in resistance (accompanied by a lesser decrease in compliance and an increase in work of breathing) which was statistically significant at the lowest concentration tested, 1.9 mg/m^3. The response was prompt in onset and resembled the pattern observed in response to sulfur dioxide. The $2.5\text{-}\mu$ particles produced a greater resistance increase at concentrations of 40 mg/m^3 than the smaller particles. This fits with the finding of Pattle *et al.*[16] that particles of 2.7μ were more lethal than those of 0.8μ when the LC_{50} was the criterion of response. On the other hand, at concentrations about 2 mg/m^3, the smaller particles produced a greater response. This underlines once again the fact that data obtained using only high concentrations can fail to predict accurately the response to low concentrations. Response to the larger particles was much slower in onset with minimal alteration during the first 15 minutes of exposure. Response to the smaller particles in 10 to 15 minutes had reached levels similar to those prevailing at the end of 1-hour exposure to the larger particles. The delayed response suggests the possibility of a different mechanism of action, and this possibility is borne out by both the mechanical behavior of the lungs and the post-mortem appearance of the lungs. In the animals exposed to the $2.5\text{-}\mu$ particles, resistance increases were accompanied by more marked compliance changes than those produced by the smaller particles or by sulfur dioxide. Such changes are consistent with the development of obstruction of major airways. At post-mortem the animals exposed to high concentrations of the large particles showed areas of atelectasis frequently involving an entire lobe. The lungs from these animals showed a lung weight to body weight ratio greater than in control animals. Such alterations were not produced by the exposures to sulfur dioxide or smaller sulfuric acid droplets. The large particles probably act by producing mucosal swelling, secretion, and exudation of fluid which leads to obstruction of major airways, whereas the smaller particles act via bronchoconstriction.

The response shown by 2 mg/m^3 (a 50 percent increase in resistance for the $0.8\text{-}\mu$ sulfuric acid) is well above the sensitivity of the method for detection of response in the guinea pig. Data on concentrations lower than this are not currently available.

The irritant potency of zinc ammonium sulfate (which may be regarded as the prototypical acid sulfate) was studied at 4 different particle sizes between 0.29μ and 1.4μ (mean size by weight).[27] Concentrations were from 0.25 mg/m^3 to 3.6 mg/m^3, and all levels tested produced an increase in flow resistance in guinea pigs. The irritant potency increased as the particle size decreased, a small increment in concentration of small particles producing a greater increment in response than did the larger particles. The importance of particle size emphasizes the inadequacy of using mass concentration data alone when attempting to assess the irritant potency of particulate material. Zinc sulfate and ammonium sulfate were also irritant but their potency was much less than that of the double salt. The authors, while not advancing an "explanation" of the Donora disaster, indicate that the zinc ammonium sulfate, zinc sulfate, and ammonium sulfate reported by Hemeon[43] as constituents of the Donora fog could have been contributors to respiratory effects if present as $0.3\text{-}\mu$ to $0.5\text{-}\mu$ particles; if the particles were 1.4μ or larger, the substances would have contributed little to the effects.

Nadel *et al.*[44] studied the effect of aerosols of histamine and zinc ammonium sulfate (as an example of an irritant aerosol) administered to anesthetized and artificially ventilated cats. The aerosol particles were smaller than 1μ and the concentration for the sulfate was 40 mg/m^3 to 50 mg/m^3. The sulfate aerosol produced a physiological response similar to that of histamine but lesser in degree. The response to 3-minute inhalation included increased pulmonary resistance, decreased pulmonary compliance, and increased end-expiratory transpulmonary pressure. Injection of atropine did not prevent the changes in compliance but decreased the changes in resistance. A bronchodilator given intravenously or as an aerosol prior to administration of the irritant prevented the changes, suggesting that they were due to smooth muscle contraction. Cardiac arrest during histamine inhalation did not prevent the changes, suggesting that histamine acts directly on the airway smooth muscle and is not dependent on the circulatory route. To correlate with the physiological responses, anatomical studies were made after rapid freezing of the lungs in the open thorax. These showed that the principal sites of constriction were the alveolar ducts and terminal bronchioles. Bronchioles up to 400μ were narrower than in control animals and showed longitudinal furrowing of the mucosa. Bronchi and bronchioles larger than 400μ were not significanty different from controls. The authors conclude: "It remains to be demonstrated that acute changes reported here at aerosol concentrations of 40 mg/m^3 to 50 mg/m^3 are exaggerated, but similar in nature to those produced, if any, at lower concentrations."

Amdur and Underhill[45] have recently reported that 1 mg/m^3 of ferric sulfate produced a 77 percent increase in flow resistance in guinea pigs, which classified it as irritant. On the other hand, neither ferrous sulfate nor manganous sulfate at this concentration produced any alteration in resistance, suggesting that the irritant potency is not related to the sulfate ion as such. In experiments on guinea pigs with sulfuric acid or particulate sulfate,[23 27 42] the response remained above control values following exposure instead of reverting to control values within an hour in the manner seen with exposure to sulfur dioxide. This difference arises since the irritant particle is cleared less rapidly from the lung than the irritant gas.

D. EFFECTS OF SULFUR DIOXIDE ON CILIARY ACTION

Cralley[46] studied the effect of irritant gases, among them sulfur dioxide, on the ciliary activity of excised rabbit trachea. Temperature and humidity were controlled and the gas to be studied was passed over the tissue at a rate comparable to that of breathing by the animal. The time needed for cessation of ciliary activity following irritant exposure was related to the concentration of irritant administered. There was a rough correlation between the concentrations of sulfur dioxide needed to produce ciliary cessation in 10 minutes in the rabbit trachea (18 ppm to 20 ppm), and the values reported for concentrations causing immediate irritation of the throat in human subjects (8 ppm to 12 ppm).

The most extensive studies of the effect of sulfur dioxide and other irritant gases on ciliary activity of experimental animals have been made by Dahamn and his co-workers, using rats[12 47] and rabits.[47-50]

The original work with rats[12 51] developed methods for measuring both rate of transport of mucus and the frequency of ciliary beat in the trachea of living animals under carefully controlled conditions of temperature and humidity. The action of sulfur dioxide on ciliary activity was tested at 50 ppm, 25 ppm, and 12 ppm (140 mg/m^3, 70 mg/m^3 and 35 mg/m^3). The time required for cessation of ciliary beat varied with the concentration, being 50 seconds at 50 ppm, 2 minutes at 25 ppm, and 4 to 6 minutes at 12 ppm. Rats which had been exposed to 10 ppm (about 30 mg/m^3) for 48 days were examined for acute response to 20 ppm (about 60 mg/m^3) to compare their response with that of control rats not previously exposed. The results indicated that the previous exposure had not altered the reactivity of the cilia to the 20-ppm exposure. In all these experiments the cilia regained mobility a few minutes after exposure ceased.

There was a marked slowing of mucus

transport and a complete cessation of flow in some rats exposed for 18 to 67 days to about 10 ppm sulfur dioxide. On the other hand, the rate of ciliary beat was slightly diminished in the shorter term exposures and unaffected in the longer exposures. These findings were readily accounted for by the morphological changes which included a thickening of the mucus blanket from 5μ to 25μ and evidence of excess secretion. The rate of transport of mucus remained depressed in animals examined a month or so after the termination of exposure, in agreement with the persistence of the observed morphological changes.

Dalhamn studied the acute effect of sulfur dioxide on ciliary activity on the trachea of the rabbit *in vivo* and *in vitro*. The *in vivo* studies were made with the animals breathing spontaneously through the nose. Concentrations of the order of 300 ppm (about 860 mg/m^3) were required to produce cessation of ciliary beat in the spontaneously breathing rabbits. Obvious explanations for the contrast of this finding with the earlier findings on rats were either that the cilia of the rabbit trachea were relatively insensitive to sulfur dioxide or that the sulfur dioxide was not reaching the site of action. Experiments with the rabbit trachea *in vitro* showed that concentrations of sulfur dioxide of 7 ppm to 10 ppm (20 mg/m^3 to 29 mg/m^3) were sufficient to produce cessation of ciliary activity. The absorption studies discussed below indicate that these findings are explicable on the basis of major absorption in the nasal cavity and pharynx of animals exposed via this route.

E. LIFETIME EXPOSURE OF ANIMALS TO SULFUR DIOXIDE

There are two brief reports [52, 53] of a study in which rats were exposed over most of their lifetime to concentrations of 1, 2, 4, 8, 16, and 32 ppm (about 3, 6, 11, 23, 46, and 92 mg/m^3) sulfur dioxide. The life span of the rats appeared to be reduced by 0.08 months for each 1 ppm increase of sulfur dioxide concentration. Statistical analysis,[54] however, fails to attach significance to these changes.

F. ABSORPTION AND DISTRIBUTION OF SULFUR DIOXIDE

Balchum *et al.*[30, 31, 33] used labelled $S^{35}O_2$ to measure the absorption, distribution, and retention of sulfur dioxide in dogs. The effect of nose and mouth breathing as compared with breathing the gas through a tracheal cannula was examined, as well as the uptake and distribution following exposure of a segment of trachea only. Following inhalation, the sulfur dioxide was widely distributed to the tissues with obviously greater amounts in the respiratory tract. A smaller porportion of the inhaled gas was found in the trachea, lungs, hilar lymph nodes, liver, and spleen when the animals breathed through the nose and mouth than when the same amount was inhaled via a tracheal cannula. The labelled $S^{35}O_2$ was only slowly removed from the trachea and lungs, and its presence in these tissues could be detected a week after exposure. Ninety percent of inhaled sulfur dioxide was removed from the airstream and became localized in the respiratory tract including the pharynx. When a tracheal segment alone was exposed to the gas, S^{35} was detected in various tissues, indicating that it can be absorbed from the upper respiratory tract alone. S^{35} was detected in the urine but not in the feces.

Bystrova [55] found a wide distribution of S^{35} throughout the tissues of rats following inhalation of high concentrations of sulfur (S^{35}) dioxide. The amount of S^{35} found in the blood and tissues was related to the concentration inhaled. The S^{35} was attached to protein and could still be found 11 days after exposure. Traces, especially in the lungs, were detectable 3 weeks aftr exposure.

Frank *et al.*[56] exposed the surgically isolated airways of the head and upper neck of anesthetized, paralyzed dogs to 22-ppm (63 mg/m^3) sulfur dioxide labelled with S^{35} and delivered at a rate of 3.5 1/min for 30 to 60 minutes. The trachea was cannulated below this region, and the lungs were ventilated with room air by a positive displacement pump. In 10 out of 12 measurements, over 95 percent of the sulfur dioxide administered to the isolated upper airways was found to be absorbed by the mucosa. In two of the animals, sampling was continued into the recovery period after the upper airways had been returned to room air, and sulfur dioxide was detected in one dog 5 to 37 minutes after exposure. In the other dog sulfur dioxide was present in the downstream sample 10 minutes after exposure but not at 60 minutes. The presence of sulfur dioxide was presumably due to desorption from the mucosa. In all 5 animals, sulfur dioxide was found in one or more expired gas samples collected either at the carina or from a bronchus. Since no sulfur dioxide entered the lower airways in inspired air, the authors conclude that the lungs were releasing the sulfur dioxide during expiration, presumably from the pulmonary capillaries. S^{35} appeared in the blood within 5 minutes of the start of exposure and continued to increase throughout the exposure. The radioactivity of the blood fell slowly during the 2-hour observation period following exposure; it was greater in the plasma than in the blood cells. Aeration of the venous blood sample caused a loss of about 20 percent of its radioactivity, but little or no radioactivity was lost by aeration of arterial blood. Radioactivity was also detected in the urine within minutes after exposure.

Dalhamn and co-workers[48–50] studied the absorptive capacity of the nasal cavity of rabbits for sulfur dioxide. Some animals breathed spontaneously through the nose from a chamber containing sulfur dioxide, and samples were taken from cannula inserted in the trachea immediately below the thyroid. In such animals, an initial exposure concentration of about 100 ppm (~290 mg/m^3) had been reduced to about 2 ppm (about 6 mg/m^3) by the time it reached the tracheal sampling tube. A concentration of about 240 ppm (about 690 mg/m^3) was reduced to about 2 ppm to 3 ppm by passage through the nasal cavity. During experiments in which air was pulled through the animal's mouth, the absorption was less. A concentration of 190 ppm (540 mg/m^3) in the inhaled air reached the trachea at about 15 ppm (about 43 mg/m^3). This observation fits with the observation by Speizer and Frank[57] that the increase in pulmonary flow resistance in human subjects was greater during mouth-breathing than during nose-breathing at a given concentration of sulfur dioxide in the inhaled air. Somewhat lower absorption values were obtained when air was sucked through the nasal cavity than had been obtained with the animals breathing spontaneously through the nose.

The overall conclusion is that at levels of 100 ppm to 300 ppm (290 mg/m^3 to 860 mg/m^3), 90 percent or more of the inhaled sulfur dioxide does not reach the lungs. This offers a reasonable explanation for the high levels that are needed in experimental animals to produce lung pathology and mortality.

Standberg[28] studied the absorption of sulfur dioxide by rabbits breathing spontaneously through the nose. A sampling device was implanted in the trachea, and the sulfur dioxide was tagged with S^{35} for analytical purposes. A concentration range of 0.05 ppm to 700 ppm (145 μg/m^3 to 2,000 mg/m^3) in the inhaled air was studied. There was considerable scatter in the data, but it was clear that at concentrations of about 100 ppm (~290 mg/m^3) there was an absorption of approximately 95 percent at inspiration and approximately 98 percent at expiration. As the concentration decreased, the absorption was less efficient, and at levels of 0.1 ppm (285 μg/m^3) and below only about 40 percent was absorbed during inspiration and about 80 percent at expiration, with some experiments showing even lower absorption efficiencies. It was proposed that the higher concentrations cause excess secretion of mucus and increase the absorption efficiency.

Amdur[24] made use of Strandberg's data to replot the dose-response curve relating resistance increase to concentration of sulfur dioxide. The data on guinea pigs were available over the range of 0.16 ppm to 835 ppm (460 μg/m^3 to 2,390 mg/m^3), which was essentially the same range covered by Strandberg's studies. If the nasal absorption was 95 percent or 98 percent for high concentrations but only 40 percent or 50 percent for low concentrations as Strandberg observed, and if the response observed resulted from the concentration reaching the lung, this could perhaps explain the nonlinearity of the dose-response curve. When the air concentrations were calculated as effective "lung concentrations" using Strandberg's data, a straight line did result. At levels below 100 ppm ($\sim$290 mg/m^3), the experimental curve for animals breathing through a tracheal cannula gave values quite close to the hypothetical "lung" curve. Strandberg's finding that, at low concentrations, sulfur dioxide penetrated the upper respiratory tract probably explains the similarity of response with and without the cannula at concentrations of 0.4 ppm (1.1 mg/m^3). Strandberg's data obtained on rabbits appear to be applicable to guinea pigs.

G. MISCELLANEOUS BIOCHEMICAL EFFECTS OF SULFUR DIOXIDE

Since experiments have demonstrated the wide distribution of S^{35} throughout the body following inhalation of sulfur dioxide, it is reasonable to search for possible biochemical effects. A biochemical lesion often is the fundamental basis for the response of an organism to toxic agents, and with many compounds the finding of such a biochemical lesion has provided the key to an understanding of their toxic action. There have been a few biochemical studies,[58–61] of animals exposed to sulfur dioxide, but from these no clear-cut picture of a biochemical lesion has emerged.

H. SUMMARY

This chapter describes animal toxicology of sulfur oxides. Both sulfur dioxide and sulfuric acid are considered in terms of dosage required to produce death, pathological change, and changes in pulmonary function.

Both sulfur dioxide and sulfuric acid irritate the respiratory system; they must, however, be employed at high concentrations if mortality is chosen as the criterion of response. Sulfuric acid is more toxic than sulfur dioxide, and its toxicity is dependent on particle size. The guinea pig is apparently the most susceptible laboratory animal studied to date, although it can withstand concentrations of sulfuric acid which would be intolerable to man.

Compared to realistic air pollutant levels, it requires relatively high concentrations of sulfur dioxide or sulfuric acid to produce pathological lung change or mortality in animals. The type of pathological change observed in the guinea pig depends on whether the death was rapid as a result of bronchial spasm or was delayed.

Sulfur dioxide is capable of producing bronchoconstriction in experimental animals such as the guinea pig, the dog, the cat, and also in man. This bronchoconstrictive property is common to various respiratory irritants. Receptors in the tracheobronchial tree have been shown to be sensitive to sulfur dioxide, and bronchoconstriction can be elicited by exposing only the upper airways to sulfur dioxide. The response is blocked by atropine or by cooling of the cervical vagosympathetic nerves.

Dose-response curves have been established for the guinea pig; they relate the concentration of sulfur dioxide to the observed increase in pulmonary flow resistance produced by one hour exposures. Slight increases in resistance are detectable at 0.16 ppm (460 mg/m^3) and the changes are readily reversible. The speed of onset (at least at high concentrations) and ready reversibility suggest that changes in smooth muscle tone are the cause of the observed bronchoconstriction.

Sulfuric acid and some, but not all, particulate sulfates also produce bronchoconstriction in the guinea pig. The response is highly dependent on particle size, with the smallest particles showing the greatest irritant potency. Comparative data are available only for the guinea pig; in this animal, sulfuric acid and irritant particulate sulfates have a greater irritant potency at a given concentration than sulfur dioxide alone.

Data obtained on guinea pigs suggest that the response to low concentrations is similar in type to that produced by higher concentrations but that the response decreases in magnitude with decreasing concentration. The nature of the experimental techniques used on guinea pigs may render the animals especially sensitive to irritant action. Increases in flow-resistance produced in these animals are not considered as indications of major physiological change and are not suitable for direct extrapolation.

The physiological response elicited in cats by small particulate irritants is probably caused by reflex action on airway smooth muscle. Morphological studies show that the principal sites of constriction are the aveolar ducts and terminal bronchioles. Bronchi and bronchioles larger than 400 μ are unaffected. The response to the particulate irritants disappeared more slowly than did the response to gaseous irritants.

To summarize our knowledge of the absorption and distribution of sulfur dioxide, it has been shown that sulfur dioxide is absorbed in the upper airways, and nasal absorption can account for as much as 95 percent to 98 percent at high sulfur dioxide concentrations. This absorption provides a protective effect to the remainder of the pulmonary system and responses are generally greater when sulfur dioxide is breathed via tracheal cannulae.

I. REFERENCES

1. Setterstrom, C. "Effects of Sulfur Dioxide on Plants and Animals." Ind. Eng. Chem., Vol. 32, pp. 473–479, April 1940.
2. Negherbon, W. O. "Sulfur Dioxide, Sulfur Trioxide, Sulfuric Acid and Fly Ash: Their Nature and Their Role in Air Pollution." Edison Electric Institute, New York, Pub.–66–900, 1966.
3. Weedon, F. R., Hartzell, A., and Setterstrom, C. "Toxicity of Ammonia, Chlorine, Hydrogen Cyanide, Hydrogen Sulphide, and Sulphur Dioxide Gases. V. Animals." Contrib. Boyce Thompson Institute, Vol. 11, pp. 365–385, Oct.–Dec. 1940.
4. McCallum, S. E. and Setterstrom, C. "Toxicity of Ammonia, Chlorine, Hydrogen Cyanide, Hydrogen Sulphide, and Sulphur Dioxide Gases. I. General Methods and Correlations." Contrib. Boyce Thompson Institute, Vol. 11, pp. 325–330, 1940.
5. Weedon, F. R. "Experimental Acute Gastric Ulcer Produced in Animals by Exposure to Sulfur Dioxide Gas." New York State J. Med., Vol. 42, pp. 620–623, 1942.
6. Setterstrom, C. and Zimmerman, P. W. "Apparatus for Studying Effects of Low Concentrations of Gases on Plants and Animals." Contrib. Boyce Thompson Institute, Vol. 9, pp. 161–169, 1938.
7. Leong, K. J., MacFarland, H. A., and Sellers, E. A. "Acute SO_2 Toxicity: Effects of Histamine Liberation." Arch. Environ. Health, Vol. 3, pp. 668–675, 1961.
8. Weedon, F. R., Hartzell, A., and Setterstrom, C. "Effects on Animals of Prolonged Exposure to Sulfur Dioxide." Contrib. Boyce Thompson Institute, Vol. 10, pp. 281–324, 1939.
9. Goldring, I. P., Cooper, P., Ratner, I. M., and Greenburgh, L. "Pulmonary Effects of Sulfur Dioxide Exposure in the Syrian Hamster." Arch. Environ. Health, Vol. 15, pp. 167–176, 1967.
10. O'Donoghue, J. C. and Graesser, F. E. "Effects of SO_2 on Guinea Pigs and Swine." Canad. J. Comp. Med. and Vet. Sci., Vol. 26, pp. 255–263, 1962.
11. Reid, L. "An Experimental Study of Hypersecretion of Mucus in the Bronchial Tree." Brit. J. Exp. Pathol., Vol. 44, pp. 437–445, Aug. 1963.
12. Dalhamn, T. "Mucus Flow and Ciliary Activity in Trachea of Healthy Rats and Rats Exposed to Respiratory Irritant Gases (SO_2, NH_3, HCHO): A Functional and Morphologic (Light Microscopic and Electron Microscopic Study), with Special Reference to Technique." Acta Physiol. Scand., Vol. 36, Supplementum No. 123, pp. 1–152, 1956.
13. Treon, J. F., Dutra, F. R., Cappel, J., Sigmon, H., and Younker, W. "Toxicity of Sulfuric Acid Mist." A.M.A. Arch. Ind. Hyg. Occup. Med., Vol. 2, pp. 716–734, 1950.
14. Mathur, K. and Olmstread, P. "Inhalation Toxicity of Sulfuric Acid Mist: Animal Experiment Study of the Problem." Thesis, Harvard School of Public Health, Boston, Massachusetts, Sept. 1948.
15. Amdur, M. O., Schulz, R. Z., and Drinker, P. "Toxicity of Sulfuric Acid Mist to Guinea Pigs." A.M.A. Arch. Ind. Hyg. Occup. Med., Vol. 5, pp. 318–329, April 1952.
16. Pattle, R. E., Burgess, F., and Cullumbine, H. "The Effects of Cold Environment and of Ammonia on the Toxicity of Sulphuric Acid Mist to Guinea Pigs." J. Pathol. Bacteriol., Vol. 72, pp. 219–232, July 1956.
17. Thomas, M. D., Hendricks, R. H., Gunn, F. D., and Critchlow, J. "Prolonged Exposure of Guinea Pigs to Sulfuric Acid Aerosol." A.M.A. Arch. Ind. Health, Vol. 17, pp. 70–80, Jan. 1958.
18. Bushtueva, K. A. "Toxicity of H_2SO_4 Aerosol." Gig. i Sanit., Vol. 22, pp. 17–22, 1957. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey. Vol. I. Translated by B. S. Levine, U.S. Dept of Commerce, Office of Technical Services, Washington, D.C., Jan. 1960, pp. 63–66.
19. Bushtueva, K. A. "Experimental Studies on the Effect of Low Oxides of Sulfur Concentrations on the Animal Organism." In: Limits of Allowable Concentrations of Atmospheric Pollutants. Book 5. V. A. Ryazanov (ed.), B. S. Levine (trans.), U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., March 1962, pp. 92–102.
20. Amdur, M. O. and Mead, J. "A Method for Studying the Mechanical Properties of the Lungs of Unanesthetized Animals. Application of the Study to Respiratory Irritants." In: Proc. 3rd National Air Pollution Symposium, Pasadena, California, April 18–20, 1955, pp. 150–159.
21. Amdur, M. O. and Mead, J. "Mechanics of Respiration in Unanesthetized Guinea Pigs." Am. J. Physiol., Vol. 192, pp. 364–368, 1958.

22. Mead, J. "Control of Respiratory Frequency." J. Appl. Physiol., Vol. 15, pp. 325–336, 1960.
23. Amdur, M. O. "The Physiological Response of Guinea Pigs to Atmospheric Pollutants." Int. J. Air Pollution, Vol. 1, pp. 170–183, Jan. 1959.
24. Amdur, M.O. "Respiratory Absorption Data and SO_2 Dose-Response Curves." Arch. Environ. Health, Vol. 12, pp. 729–732, 1966.
25. Amdur, M. O. "The Effect of Aerosols on the Response to Irritant Gases." In: Inhaled Particles and Vapors, C. N. Davies (ed.), Proc. Int. Symposium, Oxford, March 29–April 1, 1960, Pergamon Press, Oxford, 1961, pp. 281–292.
26. Murphy, S. D. "A Review of Effects on Animals of Exposure to Auto Exhaust and Some of its Components." J. Air Pollution Control Assoc., Vol. 14, pp. 303–308, 1964.
27. Amdur, M. O. and Corn, M. "The Irritant Potency of Zinc Ammonium Sulfate of Different Particle Sizes." Am. Ind. Hyg. Assoc. J., Vol. 24, pp. 326–333, July–Aug. 1963.
28. Strandberg, L. G. "SO_2 Absorption in the Respiratory Tract." Arch. Environ. Health, Vol. 9, pp. 160–166, Aug. 1964.
29. Davis, T. R. A., Battista, S. P., and Kensler, C. J. "Mechanism of Respiratory Effects During Exposure of Guinea Pigs to Irritants." Arch. Environ. Health, Vol. 15, pp. 412–419, 1967.
30. Balchum, O. J., Dybicki, J., and Meneely, G. R. "The Dynamics of Sulfur Dioxide Inhalation (Absorption, Distribution, and Retention)." A.M.A. Arch. Ind. Health, Vol. 21, pp. 564–569, June 1960.
31. Balchum, O. J., Dybicki, J., and Meneely, G. R. "Adsorption and Distribution of $S^{35}O_2$ Inhaled Through the Nose and Mouth by Dogs." Am. J. Physiol., Vol. 197, pp. 1317–1321, Dec. 1959.
32. Balchum, O. J., Dybicki, J., and Meneely, G. R. "Measurement of Pulmonary Resistance and Compliance with Concurrent Tissue Radioactive Sulfur Distribution in Dogs Inhaling a Labeled Air Pollutant: Sulfur Dioxide." Fed. Proc., Vol. 18, p. 6, March 1959.
33. Balchum, O. J., Dybicki, J., and Meneely, G. R. "Pulmonary Resistance and Compliance with Concurrent Radioactive Sulfur Distribution in Dogs Breathing $S^{35}O_2$." J. Appl. Physiol., Vol. 15, pp. 62–66, Jan. 1960.
34. Mead, J. and Collier, C. "Relation of Volume History of Lungs to Respiratory Mechanics in Anesthetized Dogs." J. Appl. Physiol., Vol. 14, pp. 660–678, 1959.
35. Salem, H. and Aviado, D. M. "Inhalation of Sulfur Dioxide. Comparative Behavior of Bronchiolar and Pulmonary Vascular Smooth Muscles." Arch. Environ. Health, Vol. 2, pp. 656–662, June 1961.
36. Yokoyama, E. and Ichikawa, K. "The Effect of Sulfur Dioxide upon Mechanical Properties of the Lungs in Dogs." Japan J. Industr. Health, Vol. 4, pp. 22–32, 1962.
37. Frank, N. R. and Speizer, F. E. "SO_2 Effects on the Respiratory System in Dogs." Arch. Environ. Health, Vol. 11, pp. 624–634, 1965.
38. Widdicombe, J. G. "Respiratory Reflexes from the Trachea and Bronchi of the Cat." J. Physiol., Vol. 123, pp. 55–70, 1954.
39. Widdicombe, J. G. "Receptors in the Trachea and Bronchi of the Cat." J. Physiol., Vol. 123, pp. 71–104, 1954.
40. Nadel, J. A., Salem, H., Tamplin, B., and Yokiwa, Y. "Mechanism of Bronchoconstriction." Arch. Environ. Health, Vol. 10, pp. 175–178, Feb. 1965.
41. Widdicombe, J. G., Kent, D. C., and Nadel, J. A. "Mechanism of Bronchoconstriction during Inhalation of Dust." J. Appl. Physiol., Vol. 17, pp. 613–616, 1962.
42. Amdur, M. O. "The Respiratory Response of Guinea Pigs to Sulfuric Acid Mist." A.M.A. Arch. Ind. Health, Vol. 18, pp. 407–414, Nov. 1958.
43. Hemeon, W. C. L. "The Estimation of Health Hazards from Air Pollution." A.M.A. Arch. Ind. Health, Vol. 11, pp. 397–402, 1955.
44. Nadel, J. A., Corn, M., Zwi, S., Ferch, J., and Graf, P. "Location and Mechanism of Airway Constriction after Inhalation of Histamine Aerosol and Inorganic Sulfate Aerosol." Proceedings, 2nd International Symposium on Inhaled Particles and Vapors, C. N. Davies (ed.), Pergamon Press, Oxford, 1966, p. 55.
45. Amdur, M. O. and Underhill, D. "The Effect of Various Aerosols on the Response of Guinea Pigs to Sulfur Dioxide." Arch. Environ. Health, Vol. 16, pp. 460–468, 1968.
46. Cralley, L. V. "The Effect of Irritant Gases upon the Rate of Ciliary Activity." J. Ind Hyg. Toxicol., Vol. 24, pp. 193–198, 1942.
47. Dalhamn, T. and Strandberg, L. "Acute Effect of Sulfur Dioxide on the Rate of Ciliary Beat in the Trachea of Rabbit, *In Vivo* and *In Vitro*, with Studies on the Absorptional Capacity of the Nasal Cavity." Int. J. Air Water Pollution, Vol. 4, pp. 154–167, Sept. 1961.
48. Dalhamn, T. "Studies on the Effect of Sulfur Dioxide on Ciliary Activity in Rabbit Trachea *In Vivo* and *In Vitro* and on the Resorptional Capacity of the Nasal Cavity." Am. Rev. Resp. Dis., Vol. 83, pp. 566–567, April 1961.
49. Dalhamn, T. and Sjoholm, J. "Studies on SO_2, NO_2, and NH_3: Effect on Ciliary Activity in Rabbit Trachea of Single *In Vitro* Exposure and Resorption in Rabbit Nasal Cavity." Acta Physiol. Scand., Vol. 58, pp. 287–291, 1963.
50. Dalhamn, T. and Strandberg, L. "Synergism between Sulphur Dioxide and Carbon Particles. Studies on Adsorption and on Ciliary Movements in the Rabbit Trachea *In Vivo*." Int. J. Air Water Pollution, Vol. 7, pp. 517–529, 1963.
51. Dalhamn, T. and Rohdin, J. "Mucous Flow and Ciliary Activity in the Trachea of Rats Exposed to Pulmonary Irritant Gas." Brit. J. Ind. Med., Vol. 13, pp. 110–113. April 1956.

52. Ball, C. O. T., Heyssel, R. M., and Balchum, O. J., Elliott, G. O., and Meneely, G. R. "Survival of Rats Chronically Exposed to Sulfur Dioxide." Physiologist, Vol. 3, p. 15, Aug. 1960.

53. "Air Pollution Conference Report." Pub. Health Reports, Vol. 75, pp. 1173–1189, Dec. 1960.

54. Goldsmith, J. R. Personal communication.

55. Bystrova, T. A. "Effects of Sulfur Dioxide Studied with the Use of Labeled Atoms." Gig. i Sanit., Vol. 20, pp. 30–37, 1957. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 1, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., pp. 89–97, Jan. 1960.

56. Frank, N. R., Yoder, R. E., Yokoyama, E., and Speizer, F. E. "The Diffusion of SO_2 from Tissue Fluids into the Lungs Following Exposure of Dogs to SO_2." Health Physics, Vol. 13, pp. 31–38, 1967.

57. Speizer, F. E. and Frank, N. R. "A Comparison of Changes in Pulmonary Flow Resistance in Healthy Volunteers Acutely Exposed to SO_2 by Mouth and by Nose." Brit. J. Indust. Med., Vol. 23, pp. 75–79, 1966.

58. Thompson, J. R. and Pace, D. M. "The Effects of Sulphur Dioxide upon Established Cell Lines Cultivated *In Vitro*." Canad. J. Biochem. Physiol., Vol. 40, pp. 207–217, 1962.

59. Navrotskii, V. K. "Effects of Chronic Low Concentration Sulfur Dioxide Poisoning on the Immuno-Biological Reactivity of Rabbits." Gig. i Sanit., Vol. 24, pp. 21–25, 1959. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 6, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., April 1961, pp. 157–163.

60. Prokhorov, Yu. D. and Rogov, A. A. "Histopathological and Histochemical Changes in the Organs of Rabbits after Prolonged Exposure to Carbon Monoxide, Sulfur Dioxide, and their Combination." Gig. i Sanit., Vol. 24, pp. 22–26, 1959. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 5, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., 1961, pp. 81–86.

61. Lobova, E. K. "Effect of Low Sulfur Dioxide Concentrations on the Animal Organism." In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 8, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., 1963, pp. 79–89.

Chapter 7

TOXICOLOGICAL EFFECTS OF SULFUR OXIDES ON MAN

Table of Contents

Page
A. INTRODUCTION 91
B. EVALUATION BY PULMONARY FUNCTION 91
  1. Sulfur Dioxide 91
    a. Short-Term Exposures 91
    b. Repeated Exposures 93
  2. Sulfuric Acid Aerosol 94
C. NASAL ADSORPTION 95
D. EFFECT ON REMOVAL OF MUCUS FROM THE RESPIRATORY TRACT 96
E. SENSORY THRESHOLD CONCENTRATIONS 96
  1. Odor Perception Threshold 96
  2. Sensitivity of the Dark-Adapted Eye 97
  3. Interruption of Alpha Rhythm 98
  4. Optical Chronaxie 98
F. SUMMARY 99
G. REFERENCES 100

List of Tables

Table
7-1 Threshold Concentrations of Sulfur Dioxide, Sulfuric Acid and their Combinations for Various Reflex Responses 99

Chapter 7

TOXICOLOGICAL EFFECTS OF SULFUR OXIDES ON MAN

A. INTRODUCTION

Reviews of the health effects of air pollution have been made by Heimann,[1] Goldsmith,[2] Lawther,[3] Phair,[4] Phillips,[5] Catcott,[6] Stokinger,[7] and Anderson.[8] In addition, a number of shorter papers report the health effects of polluted air which contains oxides of sulfur along with other pollutants.[9-27] These papers reveal the complexity of the problem of assessing the effects of air pollution. Anderson [8] states that although there are many serious limitations to published research on the health hazards of air pollution, both epidemiological and detailed clinical, physiological, and animal experiments confirm that these hazards exist. He emphasizes the importance of particulate material as a major contributor to the noted responses, or as an index of air pollution related to these effects.

This chapter considers the available evidence, reported in the literature, on the toxicological effects of the oxides of sulfur on man. More extensive reviews of the toxicology of oxides of sulfur have been presented by Greenwald [28] and Amdur.[29] The *combined* effects of sulfur dioxide and particulate matter are discussed in Chapter 8.

Much of the human toxicological research has been oriented towards occupational health. Considerations affecting community air pollution must take account of the reactions of unusually sensitive individuals, so that the published studies are generally of limited applicability to the establishment of community air quality criteria.

B. EVALUATION BY PULMONARY FUNCTION

Various investigators have examined the response of human subjects exposed for brief periods to known concentrations of sulfur dioxide or sulfuric acid mist, and measured the effects on pulmonary function. The most valuable of these studies are those dealing with constriction of the airways as reflected in measurements of pulmonary flow resistance. Such experiments evaluate directly the response of man, thus having the advantage that extrapolation from the response of some other species of animal is avoided. On the other hand, the number of individuals used in these studies is necessarily limited, and a paper reporting the response of 10 subjects is considered a major contribution. It is difficult to extend results obtained on so limited a sample to define the response of any species of animals, and no one seriously proposes such extrapolations.

1. Sulfur Dioxide

a. Short-Term Exposures

Sim and Pattle [30] exposed healthy males aged 18 to 45 to sulfur dioxide either by face mask (264 exposures) for 10 minutes or in a chamber (330 exposures) for 60 minutes. During the chamber exposures the men were allowed to walk around or smoke if they wished. Mask exposures used concentrations from about 1 ppm to 80 ppm (~3 mg/m^3 to ~230 mg/m^3), while concentrations in the chamber were from 1 ppm to 23 ppm (~3 mg/m^3 to ~65 mg/m^3). The subjects were observed clinically, and increases in airway resistance greater than 20 percent were recorded.

From the wide range of concentrations, and the large number of exposures, a dose-response relationship was found. Unfortunately, it was not expressed in as much detail as the extensive experimental data warranted on this occasion. It was reported that with dosages below 800 mg-min/m^3 (equivalent to exposures of 10 minutes to

30 ppm or 60 minutes to 5 ppm) little change was noted either clinically or by measurement of lung resistance to air flow. Occasionally, there was an increase in lung resistance sufficient to be detected by the instrument used. At the same time, there were auscultatory signs of irritation in the chest of some, but not all, of the subjects. When dosages above 1330 mg-min/m^3 (presumably 50 ppm for 10 minutes or 9 ppm for 60 minutes) were used, the lung resistance increased significantly above normal in 50 percent of the people exposed. The authors did not define "significantly above normal" in this context. Measurements made during exposure showed that the increase of airway resistance occurred within the first 10 minutes with little further change at the end of an hour, which makes the authors' choice of the $C \times t$-relationship to describe the pattern of response seem at variance with their findings. Rhinorrhea and lachrymation were common symptoms at higher dosages, but the most frequent physical finding was the presence of high-pitched musical rales, with a tendency to prolongation of the expiratory phase of respiration. Moist rales were commonly heard in the more peripheral regions of the lung after prolonged exposure, whereas in those who received smaller dosages or shorter exposures, rales were heard anteriorly over the large bronchi. Once again, this result seems at variance with the use of a $C \times t$-relationship in connection with the data.

There was no correlation between familial history of allergy and symptomatology, but in isolated instances there was evidence of increased flow resistance and considerable discomfort in those who had previously been sensitive to fog in the London area or who had definite personal histories of allergy. The effect of the smoking habits of the subjects, or the effect of smoking during exposure, were not discussed.

In 8 out of the 594 exposures there were undefined "significant changes" in pulse rate, respiratory rate, volumes of tidal or supplemental air, vital capacity, maximum breathing capacity, or blood pressure. Two of these individuals had previous personal histories of allergy and 2 had experienced previous discomfort from smog. The other 4 were suffering from the onset of mild respiratory infections on the day of exposure. One person developed a unilateral, nonspecific pleural effusion 1 week after exposure. The investigators themselves were in normal health at the start of the experiments, which extended over a 10-month period. They both developed what appeared to be an increased sensitivity to sulfur dioxide and sulfuric acid mist. One of them, who was entirely free of chest symptoms at the outset, developed a moderately severe but extremely persistent bronchitis which was immediately exacerbated into an uncomfortable period of coughing and wheezing on exposure to either sulfur dioxide or sulfuric acid.

Frank and co-workers[31-34] have examined the response of human subjects to sulfur dioxide. In the experiments reported initially,[31] 11 healthy adults were exposed on separate occasions to average sulfur dioxide concentrations of 1 ppm ($\sim$3 mg/m^3), 5 ppm ($\sim$14 mg/m^3), and 13 ppm ($\sim$37 mg/m^3). Exposures lasted 10 minutes to 30 minutes and, for each subject, were spaced at least 1 month apart. The subjects were seated in a body plethysmograph, breathing spontaneously by mouth while measurements of respiratory mechanics were made with an esophageal catheter. The measurements did not necessitate the interruption of exposure to the gas. At 1 ppm, only one of the 11 subjects showed a statistically significant increase in flow resistance, and his control resistance was the highest encountered. One other subject showed a statistically significant decrease in resistance during exposure to 1 ppm. At the 5 ppm level, 9 out of the 11 subjects showed a statistically significant increase in resistance over control values, and the average increase for the group was 39 percent above control. At 13 ppm all subjects showed an increase in resistance, and 9 of these measurements were statistically significant on an individual basis. The average increase for the group was 72 percent above control.

The data obtained in man may be compared with data obtained by similar methods in guinea pigs exposed to the same concentrations. Data for guinea pigs were available for animals breathing normally and breathing through a tracheal cannula. The human subjects were mouth-breathing. It was found that, except at the lowest gas concentration, the human subjects appeared to be slightly *more* sensitive than the guinea pigs. The most comparable dose-response relationships were for the tracheotomized guinea pigs and the normal human subjects.

The time course of the resistance changes was examined. Within 1 minute of exposure, flow resistance had increased significantly. The increase after 5 minutes was greater than the increase at 1 minute, but no further significant change occurred between 5 minutes and 10 minutes. If the 5-minute and 10-minute values are averaged to represent the "peak" response, then 45 percent of this peak was reached within the first minute of exposure. Five exposures to the higher two concentrations (5 ppm and 13 ppm) were extended to 30 minutes, but although the resistance remained elevated throughout this exposure period, the resistance tended to decrease slightly from the 10-minute values. (In this regard, the response of human subjects to sulfur dioxide differed from that of guinea pigs, in which the resistance did not decline during an exposure period of 1 hour [35, 36] and continued to increase in a limited number of animals exposed for 3 hours.) The extension of the exposure time to 30 minutes for 5 subjects exposed to 1 ppm did not produce an increase in flow resistance, suggesting that, within the limited time intervals studied, the response is related to the concentration of sulfur dioxide and not to a $C \times t$-relationship. This statement cannot obviously be extrapolated to long-term exposures.

The average flow resistance for the group still remained elevated 15 minutes after the end of exposure to both 5 ppm and 13 ppm sulfur dioxide. At 5 ppm the difference was statistically significant in 4 individual subjects; at 13 ppm it was not significant for any individual subject. In 5 subjects, periodic measurements were made within the 15-minute period and it was found that, at times, recovery may be completed within less than 5 minutes after exposure ceases. This point should be borne in mind when using techniques which involve a lapse of time between exposure and measurement.

A decrease in compliance was noted in only one single instance during an exposure to 13 ppm. At the same time the increase in flow resistance of this individual was below the average of the group. Once again the response of human subjects appears to differ from that of guinea pigs in which slight decreases in compliance appeared to accompany the resistance increase. There was a slight increase in functional residual capacity at 13 ppm, but the two lower concentrations (1 ppm and 5 ppm) produced no change. Alterations in maximum flow rate (as measured with the Wright peak flowmeter) and in timed vital capacity were minimal when compared to alterations in pulmonary flow resistance. Exposures which increased the pulmonary flow resistance by 89 percent showed only a 7 percent decrease in peak flow and, though the timed vital capacity values were generally lower, the changes expressed as percentages were minimal. A possible explanation for the relative insensitivity of these measurements is that they are preceded by a maximal inspiratory effort which may temporarily obliterate changes of flow resistance of the magnitude of those seen in this study. The smoking habits of the subjects did not appear to influence the response to sulfur dioxide.

b. Repeated Exposures

Frank *et al.*[34] also found that when sulfur dioxide (either with or without sodium chloride aerosol) is administered twice in the course of one experiment (with a 15-minute period of clean air between exposures), the response to the second exposure is less than the response to the first. The human subjects thus showed an adaptation to repeated exposure to the gas.

Speizer and Frank [33] compared the effect of sulfur dioxide on human subjects breathing the gas by nose and by mouth. Eight subjects were studied. Exposures were to either 15 ppm ($\sim$43 mg/m^3) or 28 ppm ($\sim$80 mg/m^3) sulfur dioxide and were of 10-minute duration. Pulmonary flow resistance was measured during both types of exposure. The flow resistance of the nose was measured during the nose breathing exposures. When sulfur dioxide was breathed by mouth the pulmonary flow resistance increased in 9 out of 12 experiments, and the magnitude of the change was on the average greater at the higher concentration. When the same concentrations of sulfur dioxide were administered by nose, pulmonary flow resistance increased in only 3 out of 12 experiments and decreased in one experiment. During the 15 minutes following the end of exposure the resistance remained elevated in 5 of the 12 experiments in which the gas was breathed by mouth. Four of these exposures were to 28 ppm. When the gas was breathed by nose the pulmonary flow resistance was often higher during the recovery period than it had been during exposure. In 3 experiments it rose significantly for the first time during the recovery period, and in 2 others the increase was greater during recovery than it had been during exposure.

The response of nasal resistance to sulfur dioxide was variable. In 8 of the 12 experiments it increased at some point during exposure; in 3 experiments there was a decrease; and 1 subject showed first a decrease and then an increase during the same exposure. The subjects who showed an increased nasal resistance experienced no difficulty in breathing.

When exposed by mouth, most of the subjects coughed several times during the first few minutes and had slight burning sensations of the throat and substernal area for at least 5 minutes. When exposed by nose, there was little coughing and no chest symptoms although some subjects did experience irritation of the posterior pharynx which lasted a few minutes.

Nadel *et al.*[37] reported that airway resistance, determined with a body plethysmograph, was increased by exposure to 2 ppm ($\sim$6 mg/m^3) and 5 ppm ($\sim$14 mg/^{3}) sulfur dioxide for 3 to 10 minutes. One individual out of 7 exposed to 5 ppm for 10 minutes, showed a marked increase in airway resistance which was reversible by isoproterenol inhalation. This subject had no previous history of pulmonary disease. In later studies [38] [39] an examination was made of the response of 7 subjects exposed by mouth for 10 minutes to from 4 ppm to 6 ppm sulfur dioxide. Such exposures increased the airway resistance, and the onset of change varied from 10 seconds to 4 minutes. A maximum response was usually observed within 1 minute, but in 2 subjects the resistance increased during continued exposure, and in 2 others the resistance decreased during exposure. The subject who showed the greatest change became dyspneic and wheezed during the exposure. After subcutaneous injection of 1.2 mg to 1.8 mg atropine sulfate, sulfur dioxide caused no significant change in airway resistance. Most subjects coughed and some experienced irritation of the pharynx and substernal area. These symptoms were not affected by the atropine.

Tomono [40] reported that the lowest level of sulfur dioxide which could induce bronchoconstriction in 46 healthy male subjects was 1.6 ppm ($\sim$4.6 mg/m^3). The changes were relieved by isoproterenerol inhalation.

Burton *et al.*[41] examined airway resistance and dynamic lung compliance in 10 subjects immediately following 30-minute exposures to 1 ppm ($\sim$3 mg/m^3) to 3 ppm ($\sim$9 mg/m^3) sulfur dioxide. Comparison with individual or mean group controls did not reveal significant increases in resistance or compliance during quiet breathing or during hyperventilation.

2. *Sulfuric Acid Aerosol*

Amdur *et al.*[42] exposed subjects to sulfuric acid mist of MMD 1μ at concentrations of 0.35 mg/m^3 to 5 mg/m^3 for 15 minutes. Respiration rate, tidal volume, and minute volume were measured. The subjects breathed through a pneumotachograph which permitted the measurement of inspiratory and expiratory flow rate. In 15 subjects exposed to 0.35 mg/m^3 to 0.5 mg/m^3, the respiration rate increased about 30 percent above control values, the maximum inspiratory and expiratory flow rates decreased about 20 percent, and tidal volume decreased about 28 percent. These changes occurred within the first 3 minutes of exposure and were maintained throughout the 15-minute exposure period. When the exposure ended, lung function returned rapidly to preexposure levels.

The first minute after termination of the exposure the tidal volume rose above control values and then returned to preexposure levels. Breathing through the same apparatus with omission of the acid mist was done as a control and no such changes were observed. At the highest level (5 mg/m^3) the acid mist was perceptible to all, and some subjects showed a marked response. The response was much more varied at this level, the main effect being a decrease in minute volume and a prolongation of the expiratory phase of the respiratory cycle. These experiments do not indicate whether bronchoconstriction was the response to sulfuric acid, although they suggest that this may be the case.

Sim and Pattle [30] studied the response of healthy males, aged 18 to 46, to sulfuric acid mist. They made 183 exposures of 10 minutes by mask and 316 60-minute chamber exposures to an acid mist at a relative humidity of 62 percent. The droplet size was 1μ and the strength of the sulfuric acid droplets was 10 N. They also made a total of 40 exposures in the chamber at 91 percent relative humidity. The droplet size was 1.5μ and the normality was 4 N, and concentrations of the dry mist were 3 mg/m^3 to 39 mg/m^3 and of the wet mist were 11.5 mg/m^3 to 38 mg/m^3. The reporting of the results was vague. The sulfuric acid was more irritant at high humidity, when exposure at 20.8 mg/m^3 for 30 minutes produced intense coughing which did not cease entirely throughout the exposure period. The mist was described as "almost intolerable at the onset" but the men "were able to continue for a period of 30 minutes." No airway resistance measurements could be made during the first 10 minutes. When the coughing had diminished enough to permit measurement, the increases ranged from 43 percent to 150 percent above normal. When the exposure was to dry mist at 39 mg/m^3 for a 60-minute exposure, the mist was well tolerated and there was minimal coughing. All 12 men in the group showed an increase in resistance with a range of changes from 35.5 percent to 100 percent above normal. As was mentioned previously, the investigators themselves became sensitive to both sulfur dioxide and sulfuric acid mist. One of them had his last exposure to 39.4 mg/m^3 of the dry mist, and he then exhibited chest symptoms for the remainder of the exposure and wheezed persistently for 4 days thereafter. Two subjects exposed to sulfuric acid developed long-lasting bronchitic symptoms.

Two points of value from this study apply to toxicological evaluation. One is the observed effect of high humidity on the irritant potency of sulfuric acid. The other is that, in terms of sulfur equivalent, sulfuric acid is considerably more irritant to human subjects than is sulfur dioxide.

Lawther [43] believes that experiments in the laboratory in which normal subjects have inhaled sulfuric acid mists of various particle sizes but of concentrations of approximately 1 mg/m^3 have failed to produce significant alterations in airway resistance. He mentions that certain individuals show a response to sulfur dioxide at concentrations at which the group tested as a whole did not respond and suggests that no such hypersensitivity to sulfuric acid has been recorded. These opinions are presumably based on the author's own unpublished data and are difficult to evaluate.

C. NASAL ADSORPTION

Frank and Speizer [44] [45] made measurements of the uptake and release of sulfur dioxide by the human nose. Subjects breathed through a face mask with special ports for sampling tubes. Samples could be taken within the mask within the nose 1 cm to 2 cm beyond the alae nasi, and within the oropharynx as far back as could be tolerated. The electrical conductivity method was used to determine the concentration of sulfur dioxide in samples taken during both inspiration and expiration. The concentration within the mask averaged 16 ppm ($\sim$46 mg/m^3), and exposures were of 25-minutes to 30-minutes duration. The average concentration in the nose was 13.8 ppm ($\sim$39.5 mg/m^3), representing a decrease of 14 percent. The concentration at the oropharynx was too small to be measured accurately. During exhalation, the sample from the nose contained an average of 2 ppm, suggesting a desorption from the mucosa during exhalation.

D. EFFECT ON REMOVAL OF MUCUS FROM THE RESPIRATORY TRACT

In a study[46] of the effect of sulfur dioxide on the rate of removal of mucus from the respiratory tract of human subjects, exposures to from 10 ppm to 15 ppm ($\sim$29 mg/m^3 to $\sim$43 mg/m^3) for 1 hour produced a 10-percent to 15-percent decrease in the clearance rate, while a concentration of 25 ppm to 30 ppm ($\sim$72 mg/m^3 to $\sim$86 mg/m^3) caused a 45-percent to 50-percent decrease and 50 ppm to 55 ppm ($\sim$140 mg/m^3 to $\sim$160 mg/m^3) caused a 65-percent to 70-percent decrease. It was not reported whether or not the subjects were nose-breathing during exposure.

E. SENSORY THRESHOLD CONCENTRATIONS

The kinds of response to sulfur dioxide discussed in this section, although not strictly within the realm of toxicology, are appended here because the data are closely related to the discussion in this Chapter.

Many recent investigations in Russia have been directed toward determining sulfur oxides threshold concentrations for various sensory responses. These investigations have included determination of odor thresholds and the effects of sulfur dioxide on optical chronaxie, sensitivity of the dark-adapted eye to light, interruption of the alpha (α) rhythm in electroencephalograms, and interference with cortical conditioned reflexes as shown by electroencephalograms. Most of these investigations have been summarized, and the more recent methodology described, by Ryazanov.[47]

1. Odor Perception Threshold

An odor threshold is typically determined in a well-ventilated chamber containing 2 orifices from which emerge 2 small streams of gas, one very pure air and the other being a stream of the test gas. The subject sits in front of the apparatus, sniffs both orifices, and points out the odorous one. This experiment is repeated with the same concentration of test gas over a period of several days. The experiment is performed with increasingly reduced concentrations until the subject, in the majority of instances, denies the presence of an odor or gives erroneous answers. The threshold concentration for the most sensitive subject in a group of volunteers is defined as the threshold for odor perception.[47]

Using the 2-orifice apparatus described above, Dubrovskaya[48] conducted sulfur dioxide odor perception threshold tests on 12 subjects. Sulfur dioxide concentrations of 0.5 mg/m^3 to 13 mg/m^3 (0.17 ppm to 4.6 ppm) were used in 530 threshold determinations. Six test subjects sensed the odor of sulfur dioxide in the range 2.6 mg/m^3 to 3.0 mg/m^3; four subjects sensed the odor in the range 1.6 mg/m^3 to 2.0 mg/m^3; one sensed the odor in the range 2.1 mg/m^3 to 2.5 mg/m^3; and one sensed the odor in the range 3.1 mg/m^3 to 3.6 mg/m^3. Thus, the average sulfur dioxide odor threshold concentration was 0.8 ppm to 1 ppm ($\sim$2.3 mg/m^3 to $\sim$2.9 mg/m^3), and for the more sensitive of these persons it was 0.5 ppm to 0.7 ppm ($\sim$15 mg/m^3 to $\sim$20 mg/m^3); it should be noted, however, that most of the subjects were of an age at which odor perception was presumed to be most sensitive.

Bushtueva[49] reported that among 10 test subjects the minimum concentration of sulfuric acid aerosol (particle size not given) which was sensed by odor ranged from 0.6 mg/m^3 to 0.85 mg/m^3 (average 0.75 mg/m^3). In tests made on five subjects[50] a combination of sulfur dioxide at 1 mg/m^3 (0.35 ppm) and sulfuric acid mist at 0.4 mg/m^3 was below the odor threshold.

Popov *et al.*[51] used an apparatus in which the sulfur dioxide concentration could be changed rapidly and showed that the odor of sulfur dioxide could be detected at concentrations of 4 mg/m^3 (1.4 ppm). At 4.0 mg/m^3 to 6.5 mg/m^3 (1.4 ppm to 2.3 ppm), the majority of their test subjects perceived the gas as a strong odor; a few perceived it as a faint odor. Subjects described concentrations of sulfur dioxide above 8.5 mg/m^3 (3.0 ppm) as having a very sharp odor. The number of subjects involved in these studies was not stated.

Recent determination of sulfur dioxide odor thresholds[52] conducted for the Manufacturing Chemists' Association gave somewhat lower values than those cited above. The concentrations at which first one-half and then all of the panel members could positively recognize the odor were reported both to be 0.47 ppm (1.3 mg/m^3). The details of the test procedure are thoroughly discussed in the report, but one important aspect is reiterated as a reminder that odor thresholds usually represent values derived under ideally suited conditions and with trained individuals. The investigators, who were highly qualified to judge on the basis of substantial experience with consumer evaluation of known flavor and odor situations, derived threshold values, in test rooms under ideal conditions, lower than those which would be recognized by the majority of a population under ordinary atmospheric conditions. This does not mean that normal individuals exposed to sulfur dioxide under ideal test conditions could not perceive the 0.47 ppm level indicated, but because of background odor and lack of awareness or concern with ambient odor conditions, such individuals in an everyday situation would probably be less responsive to this low concentration than are subjects undergoing a test.

2. Sensitivity of the Dark-Adapted Eye

The sensitivity of the eye to light while a subject is in darkness increases with time. Several investigations have been made of the effects of inhalation of sulfur oxides on this sensitivity. Typically, measurements of a subject's normal sensitivity are taken in a dark, well-ventilated chamber in complete silence (sudden stimuli, including noise, may change the sensitivity). Each subject is tested once daily following preliminary conditioning at a high light level. Light sensitivity is measured at 5-minute or 10-minute intervals, and a normal curve of increasing sensitivity to light is established from measurements taken over a period of 7 days to 10 days.[47]

Dubrovskaya[48] studied the effect of inhaling sulfur dioxide in concentrations from 0.96 mg/m^3 to 19.2 mg/m^3 for 15 minutes before measuring light sensitivity during dark adaptation. She reported that light sensitivity was increased by sulfur dioxide concentrations of 0.96 mg/m^3 to 1.8 mg/m^3 (0.34 ppm to 0.63 ppm), that the increase in sensitivity reached a maximum at concentrations of 3.6 mg/m^3 to 4.8 mg/m^3 (1.3 ppm to 1.7 ppm), and that further increases in the sulfur dioxide concentration resulted in progressive lowering of eye sensitivity to light until at 19.2 mg/m^3 the sensitivity was identical with that of the unexposed subject.

In exposures during light adaptation, sulfur dioxide concentrations of 0.6 mg/m^3 to 7.2 mg/m^3 (0.21 ppm to 2.5 ppm) caused slight increases in eye sensitivity. Maximum sensitivity was attained at 1.5 mg/m^3 (0.52 ppm); at higher concentrations the increased sensitivity began to abate. Two human subjects were used in these experiments. The odor threshold was between 2.5 mg/m^3 and 3.0 mg/m^3 for one subject and between 3.0 mg/m^3 and 3.6 mg/m^3 for the other, so that changes in sensitivity to light during dark adaptation were caused by sulfur dioxide concentrations below the odor threshold.

Bushtueva[49] studied the effect of sulfuric acid mist on the sensitivity to light of two test subjects. The test periods were 60, 90, and 120 minutes. During the first half hour, sensitivity was measured every 5 minutes, and after that every 10 minutes. In each subject a control curve was established by 7 repeated tests, and then the effect on light sensitivity of sulfuric acid aerosol exposure for 4 minutes and for 9 minutes at the 15th and 60th minutes, respectively, was determined. With sulfuric acid mist of undetermined particle size at a concentration of 0.6 mg/m^3, a just detectable increase in light sensitivity was found as a result of the exposure at the 15th minute, but no detectable effect was observed as a result of the exposure at the 60th minute. Concentrations in the range of 0.7 mg/m^3 to 0.96 mg/m^3 brought about a well-defined increase in light sensitivity. With 2.4 mg/m^3, increased sensitivity to light was elicited by the exposures at both the 15th and 60th minutes of the test; normal sensitivity was restored in 40 to 50 minutes.

Bushtueva[50] studied the effect of sulfur dioxide, sulfuric acid mist and combinations of the two on sensitivity of the eye to light in 3 subjects. The combination of sulfur dioxide at 0.65 mg/m^3 (0.23 ppm) with sulfuric acid mist at 0.3 mg/m^3 resulted in no change in sensitivity of the eye to light. An increase of approximately 25 percent in light sensitivity resulted from exposure to either sulfur dioxide at 3 mg/m^3 (~1.0 ppm) or sulfuric acid mist at 0.7 mg/m^3. The combination of sulfur dioxide at 3 mg/m^3 with sulfuric acid mist at 0.7 mg/m^3 resulted in an increase of approximately 60 percent in light sensitivity. Exposures lasted for 4½ minutes.

3. Interruption of Alpha Rhythm

The electroencephalogram is a composite record of the electrical activity of the brain recorded as the difference in electrical potential between 2 points on the head. In the adult, the electroencephalogram characteristically shows a fairly uniform frequency from 8 cycles to 12 cycles per second in the posterior head regions. Variations occur with age, and the state of wakefulness and attentiveness, or as a result of incoming sensory stimuli from exteroceptive or interoceptive receptors. The dominant frequency is inhibited or attenuated by eye opening and by mental activity.[53]

Subjects with well-defined α-rhythms studied in a silent and electrically-shielded chamber show a temporary attenuation of the α-rhythm each time they are given a light signal. When the light is excluded, the α-rhythm returns to normal. A concentration of test gas is determined which is so low that by itself it does not cause attenuation of the α-rhythm. A subject breathes the gas at this concentration, and then he receives the light signal. After exposure to this sequence (gas then light) several times (5 to 30 times in 1 day), a subject will show attenuation *before* he receives the light signal; that is, he responds to the unperceived odor. The unperceived odor thus becomes the conditioning stimulus and brings about the so-called conditioned electrocortical reflex.[47]

Bushtueva *et al.*[54] reported that 20-second exposures of 6 human subjects to sulfur dioxide concentrations from 0.9 mg/m^3 to 3 mg/m^3 (~0.3 ppm to ~1.0 ppm) produced attenuation of the α-wave lasting 2 to 6 seconds; at concentrations of 3.0 mg/m^3 to 5.0 mg/m^3 (~1.0 ppm to 1.7 ppm) attenuation lasted throughout the 20-second exposure. Exposures to 0.6 mg/m^3 (~0.2 ppm) did not cause attenuation of the α-wave. Exposures to sulfuric acid mist at 0.6 mg/m^3 to 0.75 mg/m^3 caused attenuation of the α-wave, whereas exposures to 0.4 mg/m^3 to 0.5 mg/m^3 did not. For both substances, the threshold for attenuation of the α-wave is the same as the odor threshold or the threshold of irritation of the respiratory tract. In other experiments, Bushtueva demonstrated that electrocortical conditioned reflexes could be developed with sulfur dioxide at 0.6 mg/m^3 (~0.2 ppm) or with sulfuric acid mist at 0.4 mg/m^3, but not with lesser concentrations of either substance. Finally, Bushtueva [55] demonstrated that combinations of sulfur dioxide at 0.50 mg/m^3 (0.17 ppm) with sulfuric acid mist at 0.15 mg/m^3 or sulfur dioxide at 0.25 mg/m^3 (0.087 ppm) with sulfuric acid mist at 0.30 mg/m^3 could produce electrocortical conditioned reflexes.

4. Optical Chronaxie

Chronaxie is defined as the time required for excitation of a nervous element by a definite stimulus. In the determination of optical chronaxie, a weak electrical current is applied to the eyeball to give the sensation of a light flash. For each subject there is an intensity of stimulation (measured in volts) below which no sensation of light takes place. The time required for this minimal voltage to produce the sensation of light in a subject is the optical chronaxie for the subject. According to Pavlovian theory, the excitation of one area of the cerebral cortex may inhibit the excitation of other areas through the rule of induction.[47] It has therefore been postulated that excitation of the olfactory sensory area by the oxides of sulfur inhibits the light-sensing area of the cerebral cortex and thus increases optical chronaxie.

Bushtueva [50] studied the effects of different concentrations of sulfur dioxide, sulfuric acid mist, and combinations of the two on the optical chronaxie of three subjects. Optical chronaxie was determined in each test subject at 3-minute intervals as follows: at the start and on the 3rd, 6th, 9th, 12th and 15th minutes. Between the 6th and 9th minutes the subjects inhaled sulfur dioxide, sulfuric acid mist, or their combination for 2 minutes. In each subject the threshold concentrations of sulfur dioxide and sulfuric acid mist were first determined independently, and then threshold concentrations for combinations of the two were determined.

Results presented for one subject were:

1. Neither sulfur dioxide at 900 μg/m^3 nor sulfuric acid mist at 600 μg/m^3 produced increased optical chronaxie, but the combination of these concentrations produced a 16 percent increase in optical chronaxie;
2. concentrations of sulfur dioxide at 1200 μg/m^3 with sulfuric acid mist at 400 μg/m^3 produced no increase in optical chronaxie; and
3. concentrations of either sulfur dioxide at 1500 μg/m^3 or sulfuric acid mist at 750 μg/m^3 increased optical chronaxie, and the effects of the combination at these concentrations were additive. Similar results were reported for the other subjects.

The data obtained by Bushtueva[55] are summarized in Table 7-1. Consideration of the levels noted provides information which may be relevant to the establishment of "Level I" and "Level II" of the World Health Organization's "guides to air quality."[56] These "guides," equivalent in usage to our term "criteria," covers sets of concentrations and exposure times at which specified types of effects are noted or at which no effect is noted. The definitions of these levels are as follows:

Level I (Range I) Concentrations and exposure times at or below which, according to present knowledge, neither direct nor indirect effects (including alteration of reflexes or of adaptive or protective reactions) have been observed.

Level II (Range II) Concentrations and exposure times at and above which there is likely to be irritation of the sensory organs, harmful effects on vegetation, visibility reductions, or other adverse effects on the environment.

Level III (Range III) Concentrations and exposure times at and above which there is likely to be impairment of vital physiological functions or changes that may lead to chronic diseases or shortening of life.

Level IV (Range IV) Concentrations and exposure times at and above which there is likely to be acute illness or death in susceptible groups of the population.

The practical ramifications of these neuro-physiological responses have not been explored. In addition, one should be cautioned in the interpretation of these data since there have been no replicate studies to confirm the reported neuro-physiological responses and information concerning the experimental conditions was less than adequate.

F. SUMMARY

Various animal species, including man, respond to sulfur dioxide by bronchoconstric-

Table 7-1. THRESHOLD CONCENTRATIONS OF SULFUR DIOXIDE, SULFURIC ACID, AND THEIR COMBINATIONS FOR VARIOUS RESPONSES[55]

Procedure Used	Threshold Concentration			
	H_2SO_4	SO_2	H_2SO_4 +	SO_2
	μg/m^3	μg/m^3	μg/m^3	μg/m^3
Threshold Concentration of Irritation Effects and Odor Perception	600–850	1600–2600	>300	500
Data Obtained by the Method of Eye Adaptation to Darkness	630–730	920	>300	500
Data Obtained by the Method of Optical Chronaxie	730	1500	600	1200
Encephalographic Method	630	900	>300	500
"Electrocortical" Conditioned Reflex	400	600	150	500
			300	250

tion, which may be assessed in terms of a slight increase in airway resistance. Normal individuals, exposed to sulfur dioxide *via* the mouth, exhibit small changes in airway resistance which are often insufficient to produce any respiratory symptoms. The effects may be even smaller when the subject breathes through his nose.

Laboratory observations of respiratory irritations suggest that most individuals will show a response to sulfur dioxide at concentrations of 5 ppm (~14 mg/m^3) and above. At concentrations of 1 ppm to 2 ppm (~3 mg/m^3 to ~6 mg/m^3) an effect can be detected only in certain sensitive individuals, and on occasion, exposures to 5 ppm to 10 ppm (~14 mg/m^3 to ~30 mg/m^3) have been shown to cause severe bronchospasm in such persons; no further special study at lower concentrations has been carried out with these individuals. The exposure of the more sensitive individuals to 1 ppm (~3 mg/m^3), although it does not produce severe bronchospasm, does elicit a detectable response.

Sulfuric acid is a much more potent irritant to man than is sulfur dioxide, and its effects are highly dependent on particle size. Insufficient data are available for quantitative assessment of the health hazard. There is inadequate information on the response of human subjects to any of the other particulate sulfates. Nasal absorption and desorption of oxides of sulfur, and the effect of the oxides on the removal of mucus from the respiratory tract are briefly considered.

In most of the studies discussed, an increase in pulmonary flow resistance was the indicator of response employed. However, the concentrations of oxides of sulfur just needed to elicit certain sensory responses are presented, although the practical ramifications of these neurophysiological responses have not been fully explored. These values may ultimately have significance for the establishment of Levels I and II of air quality as promulgated by the World Health Organization.

G. REFERENCES

1. Heimann, H. "Effects of Air Pollution on Human Health." In: Air Pollution, World Health Organization Monograph Series 46, Columbia Univ. Press, New York, 1961, pp. 159–220.
2. Goldsmith, J. R. "Effects of Air Pollution on Humans." In: Air Pollution, 2nd edition, Vol. 1, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 547–615.
3. Lawther, P. J., Martin, A E., and Wilkins, E. T. "Epidemiology of Air Pollution." World Health Organization, Pub. Health Paper 15, Geneva, 1962.
4. Phair, J. J. "The Epidemiology of Air Pollution." In: Air Pollution Handbook, P. L. Magill, F. R. Holden, and C. Ackley (eds.), McGraw-Hill, New York, 1956, pp. 7–1 to 7–14.
5. Phillips, P. H. "The Effects of Air Pollutants on Farm Animals." In: Air Pollution Handbook, P. L. Magill, F. R. Holden, and C. Ackley (eds.), McGraw-Hill, New York, 1956, pp. 8–1 to 8–12.
6. Catcott, E. J. "Effects of Air Pollution on Animals." In: Air Pollution, World Health Organization Monograph Series 46, Columbia Univ. Press, New York, 1961, pp. 221–231.
7. Stokinger, H. E. and Coffin, D. L. "Biological Effects of Air Pollutants." In: Air Pollution, Vol. 1, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 445–546.
8. Anderson, D. O. "The Effects of Air Contamination on Health, Parts I, II, and III." Canadian Med. Assn. J., Vol. 97, pp. 528–536, 585–593, 802–806, 1967.
9. Roberts, A. "Air Pollution and Bronchitis." Am. Rev. Res. Dis., Part I, Vol. 80, pp. 582–584, Oct. 1959.
10. Ascher, L. "Smoke-Nuisance in Large Towns." Eng. News, Vol. 58, pp. 434–435, Oct. 24, 1907.
11. "Smog and Disease." Lancet, Vol. 267, pp. 1163–1164, Dec. 4, 1954.
12. "The Lethal Aerosol." Lancet, Vol. 265, p. 976, Nov. 7, 1953.
13. "Our Natural Element." Lancet, Vol. 265, pp. 765–766, Oct. 10, 1953.
14. Drinker, P. "Atmospheric Pollution." Ind. Eng. Chem., Vol. 31, pp. 1316–1320, 1939.
15. Laidlaw, S. A. "The Effects of Smoke Pollution on Health." J. Institute Fuel, Vol. 27, pp. 96–99, Feb. 1954.
16. Andelman, S. L. "Air Pollution, the Respiratory Tract and Public Health." Eye, Ear, Nose and Throat Monthly, Vol. 39, pp. 961–964, Dec. 1960.
17. Farber, S. M. and Wilson, R. H. L. "Air Contamination: A Respiratory Hazard." J. Amer. Med. Assoc., Vol. 180, pp. 362–366, May 5, 1962.
18. Goldsmith, J. R. "Effects of Air Pollution on Man." Connecticut Med., Vol. 27, pp. 455–464, Aug. 1963.
19. Barnes, J. M. "Mode of Action of Some Toxic Substances. With Special Reference to the Effects of Prolonged Exposure." Brit. Med. J., Vol. 2, pp. 1097–1104, Oct. 28, 1961.
20. Nelson, H. W. and Lyons, C. J. "Sources and Control of Sulfur-Bearing Pollutants." J. Air Pollution Control Assoc., Vol. 7, pp. 187–193, Nov. 1957.

21. Prindle, R. A. and Landau, E. "Health Effects from Repeated Exposures to Low Concentrations of Air Pollutants." Pub. Health Reports, Vol. 77, pp. 901–908, Oct. 1962.
22. Catteral, M. "Air Pollution, the Human Problem." Smokeless Air, Vol. 39, pp. 142–148, 1963.
23. Bell, A. "The Air We Breathe." Med. J. Australia, Vol. 44, pp. 897–921, June 15, 1957.
24. Oswald, N. C. "Physiological Effects of Smog." Roy. Meteorol. Soc. J., Vol. 80, pp. 271–278, 1954.
25. Anderson, R. J. "Epidemiologic Studies of Air Pollution." Diseases of the Chest, Vol. 42, pp. 474–481, Nov. 1962.
26. Cooper, W. C. "Epidemiologic Studies on Air Pollution." Amer. Med. Assoc., Arch. Ind. Health, Vol. 15, pp. 177–180, 1957.
27. Wallace, A. S. "Mortality from Asthma and Bronchitis in the Auckland 'Fumes Area'." New Zealand Med. J., Vol. 56, pp. 242–249, 1957.
28. Greenwald, I. "Effects of Inhalation of Low Concentrations of Sulfur Dioxide Upon Man and Other Mammals." Amer. Med. Assoc., Arch, Ind. Hyg. and Occup. Med., Vol. 10, pp. 455–475, Dec. 1954.
29. Amdur, M. O. "Report on Tentative Ambient Air Standards for Sulfur Dioxide and Sulfuric Acid." Ann. Occup. Hyg., Vol. 3, pp. 71–83, Feb. 1961.
30. Sim, V. M. and Pattle, R. E. "Effect of Possible Smog Irritants on Human Subjects." J. Amer. Med. Assoc., Vol. 165, pp. 1908–1913, Dec. 14, 1957.
31. Frank, N. R., Amdur, M. O., Worcester, J., and Whittenberger, J. L. "Effects of Acute Controlled Exposure to SO_2 on Respiratory Mechanics in Healthy Male Adults." J. Appl. Physiol., Vol. 17, pp. 252–258, March 1962.
32. Frank, N. R. "Studies on the Effects of Acute Exposure to Sulfur Dioxide in Human Subjects." Proc. Roy. Soc. Med., Vol. 57, pp. 1029–1033, 1964.
33. Speizer, F. E. and Frank, N. R. "A Comparison of Changes in Pulmonary Flow Resistance in Healthy Volunteers Acutely Exposed to SO_2 by Mouth and by Nose." Brit. J. Ind. Med., Vol. 23, pp. 75–79, 1966.
34. Frank, N. R., Amdur, M. O., and Whittenberger, J. L. "A Comparison of the Acute Effects of SO_2 Administered Alone or in Combination with NaCl Particles on the Respiratory Mechanics of Healthy Adults." Int. J. Air Water Pollution, Vol. 8, pp. 125–133, 1964.
35. Amdur, M. O. "The Effect of Aerosols on the Response to Irritant Gases." In: Inhaled Particles and Vapors. C. N. Davies (ed.), Proc. Intern Symposium, Oxford, March 29–April 1, 1960, Pergamon Press, Oxford, 1961, pp. 281–292.
36. Amdur, M. O. "The Physiological Response of Guinea Pigs to Atmospheric Pollutants." Intern. J. Air Pollution, Vol. 1, pp. 170–183, Jan. 1959.
37. Nadel, J. A., Tierney, D. F., and Comroe, J. H. "Pulmonary Responses to Aerosols." Proc. 3rd Air Pollution Med. Res. Conference, California State Dept. of Public Health, Los Angeles, California, Dec. 9, 1959, pp. 66–74.
38. Nadel, J. A., Salem, H., Tamplin, B., and Tokiwa, Y. "Mechanism of Bronchoconstriction." Arch. Environ. Health, Vol. 10, pp. 175–178, Feb. 1965.
39. Nadel, J. A., Salem, H., Tamplin, B., and Tokiwa, Y. "Mechanism of Bronchoconstriction During Inhalation of Sulfur Dioxide." J. Appl. Physiol., Vol. 20, pp. 164–167, 1965.
40. Tomono, Y. "Effects of SO_2 on Human Pulmonary Functions." Japan J. Ind. Health, Vol. 3, pp. 77–85, Feb. 1961.
41. Burton, G. G., Corn, M., Gee, J. B. L., Vassallo, D., and Thomas, A. "Absence of 'Synergistic Response' to Inhaled Low Concentration Gas-Aerosol Mixtures in Healthy Adult Males." (Presented at 9th Annual Air Pollution Medical Research Conference, Denver, Colorado, July 1968.)
42. Amdur, M. O., Silverman, L., and Drinker, P. "Inhalation of Sulfuric Aicd Mist by Human Subjects." Amer. Med. Assoc. Arch. Ind. Hyg. Occup. Med., Vol. 6, pp. 306–313, Oct. 1952.
43. Lawther, P. J. "Compliance with the Clean Air Act. Medical Aspects." J. Inst. Fuel, Vol. 36, pp. 341–344, Aug. 1963.
44. Frank, N. R. and Speizer, F. E. "Uptake and Release of SO_2 by the Human Nose." Physiol., Vol. 7, p. 132, Aug. 1964.
45. Speizer, F. and Frank, N.R. "The Uptake and Release of SO_2 by the Human Nose." Arch. Environ. Health, Vol. 12, pp. 725–728, 1966.
46. Cralley, L. V. "The Effect of Irritant Gases Upon the Rate of Ciliary Activity." J. Ind. Hyg. & Toxicol., Vol. 24, pp. 193–198, 1942.
47. Ryazanov, V. A. "Sensory Physiology as Basis For Air Quality Standards." Arch. Environ. Health, Vol. 5, pp. 479–494, Nov. 1962.
48. Dubrovskaya, F. I. "Hygienic Evaluation of Pollution of Atmospheric Air of a Large City with Sulfur Dioxide Gas." In: Limits of Allowable Concentrations of Atmospheric Pollutants, Book 3, Translated by B. S. Levine, U.S. Dept. of Commerce. Office of Technical Services, Washington, D.C., 1957, pp. 37–51.
49. Bushtueva, K. A. "The Determination of the Limit of Allowable Concentration of Sulfuric Acid in Atmospheric Air." In: Limits of Allowable Concentrations of Atmospheric Pollutants, Book 3, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., 1957, pp. 20–36.
50. Bushtueva, K. A. "Threshold Reflex Effect of SO_2 and Sulfuric Acid Aerosol Simultaneously Present in the Air." In: Limits of Allowable Concentrations of Atmospheric Pollutants, Book 4, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., Jan. 1961, pp. 72–79.

51. Popov, I. N., Cherkasov, Ye. F., and Trakhtman, O. L. "Determination of Sulfur Dioxide Odor Threshold Concentration." Gig. i Sanit., Vol. 5, pp. 16-20, 1952. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 3, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., May 1960, pp. 102–106.

52. "Determination of Odor Thresholds for 53 Commercially Important Organic Compounds." Report by Arthur D. Little, Inc. to the Manufacturing Chemists' Association, Jan. 11, 1968, 21 pp.

53. Grollman, A. (ed.) "The Functional Pathology of Disease; The Physiologic Basis of Clinical Medicine." 2nd edition, McGraw-Hill, New York, 1963, 979 pp.

54. Bushtueva, K. A., Polezhaev, E. F., and Semenenko, A. D. "Electroencephalographic Determination of Threshold Reflex Effect of Atmospheric Pollutants." Gig. i Sanit., Vol. 25, pp. 54–61, 1960. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 7, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., 1962, pp. 137–142.

55. Bushtueva, D. A. "New Studies of the Effect of Sulfur Dioxide and of Sulfuric Acid Aerosol on Reflex Activity of Man." In: Limits of Allowable Concentrations of Atmospheric Pollutants. Book 5, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., March 1962, pp. 86–92.

56. "Atmospheric Pollutants." World Health Organization, Technical Report Series 271, Geneva, 1964.

BLANK PAGE

Chapter 8

COMBINED EFFECTS OF EXPERIMENTAL EXPOSURES TO SULFUR OXIDES AND PARTICULATE MATTER ON MAN AND ANIMALS

Table of Contents

Page
A. INTRODUCTION 105

B. EVALUATION BY MORTALITY AND PATHOLOGY IN ANIMALS 105
1. Sulfur Dioxide, Sulfuric Acid, and Particles 105
2. Sulfur Dioxide and Sulfuric Acid 106

C. EVALUATION BY PULMONARY FUNCTION IN ANIMALS 107

D. EVALUATION BY PULMONARY FUNCTION IN MAN 109

E. SUMMARY 111

F. REFERENCES 111

Chapter 8

COMBINED EFFECTS OF EXPERIMENTAL EXPOSURES TO SULFUR OXIDES AND PARTICULATE MATTER ON MAN AND ANIMALS

A. INTRODUCTION

The problem of interactions of irritant gases and particulate material is one of greatest interest in air pollution toxicology. Its implications are far broader than the present specific discussion of the effect of particulate material on the response to sulfur dioxide. For a treatment of the broader aspects of the problem, see Chapters 9 and 10 in the companion document, *Air Quality Criteria for Particulate Matter.*

B. EVALUATION BY MORTALITY AND PATHOLOGY IN ANIMALS

1. Sulfur Dioxide, Sulfuric Acid, and Particles

Schnurer[1] exposed rabbits and rats (23 hours per day for 80 days) and mice (17 days) to atmospheres produced from burning equal amounts of anthracite coal, of coke, and of bituminous coal. The resulting sulfur dioxide concentrations were 1.91 ppm (5.46 mg/m^3) for anthracite coal, 9.12 ppm (26.1 mg/m^3) for coke, and 7.51 ppm (21.5 mg/m^3) for bituminous coal. The corresponding particle concentrations were 312, 370, and 4,410 particles per cc. The control atmosphere contained 125 particles per cc. Taking the weight gain of control animals as 100 percent, the rats exposed to anthracite smoke gained 105 percent, the rats exposed to coke smoke gained 114 percent, and the rats exposed to bituminous coal smoke gained 75 percent. With the rabbits these values were 84 percent, 77 percent, and 9 percent respectively. The hemaglobin percentage and the red and white blood cell counts rose in all groups, but this rise was less pronounced in the rabbits exposed to anthracite smoke. The greatest changes in the above measurements, the greatest number of uncomplicated pneumonias, and the greatest incidence of bronchitis were observed in the animals exposed to bituminous coal smoke. No significant pathological change could be detected in the lungs of animals exposed to coke or anthracite smoke 2½ months or 14 months after exposure. Animals exposed to bituminous coal smoke developed evidence of fibrosis, proliferation of the bronchial epithelium, and marked peribronchial lymphoid hyperplasia.

The exposures to anthracite coal smoke and coke smoke were essentially equivalent in terms of particle concentration, but the smoke from the coke contained about 9 ppm (~26 mg/m^3) sulfur dioxide as opposed to about 2 ppm (~6 mg/m^3) for the anthracite coal smoke. Neither of these exposures produced pathological alterations in the lungs. The smoke from coke and bituminous coal contained about 9 ppm (~26 mg/m^3) and 8 ppm (~23 mg/m^3) sulfur dioxide, but the particle concentration of the bituminous coal smoke was over 10 times that of the smoke from the coke. The bituminous smoke produced pathological alterations which were not observed in animals exposed to the smoke from the coke. The pathological alterations observed in the animals exposed to bituminous coal smoke may possibly be associated with the simultaneous presence of high levels of sulfur dioxide and smoke. Unfortunately, however, the experiments do not allow a simple interpretation.

Susceptibility to infection by pneumococci of rats exposed to coal dust or smoke has been studied.[2] Sulfur dioxide concentrations in the coal smoke ranged between 0.7 ppm (2.0 mg/m^3) and 1.6 ppm (4.6 mg/m^3), and smoke concentrations would have been considered as "dense" on visual inspection (equivalent to about a No. 3 reading on the Ringelmann chart). Exposures ranged from 2 to 154 days, and no difference was noted between the control animals and exposed animals in regard to mortality or susceptibility to infection. On the other hand, the strain of rats used normally are known to develop areas in the lung which are filled with mucus or pus, and the incidence of this condition was 20 percent in the control animals and 40 percent in animals exposed to smoke for more than 20 weeks.

Pattle and Burgess [3] studied the effect of mixtures of sulfur dioxide and smoke on mice and guinea pigs. The concentrations of sulfur dioxide used were in the range of 2,700 mg/m^3 to 12,000 mg/m^3 (940 ppm to 4,200 ppm) and the smoke concentrations were in the range of 50 mg/m^3 to 135 mg/m^3. The end point was the dosage required to produce death. With concentrations of this magnitude, the results obtained have little applicability to air pollution criteria. Although they found that the lethality of mixtures of sulfur dioxide and smoke was greater than the lethality of the sulfur dioxide alone, they considered the effect to be a simple additive one resulting from the action of smoke in blocking the bronchi and alveoli.

Salem and Cullumbine [4] studied the effects of kerosene smoke on the acute toxicity to guinea pigs and mice of various irritants, among them sulfur dioxide and sulfuric acid. As in the work of Pattle and Burgess,[3] the concentrations were orders of magnitude greater than those found in polluted atmospheres. Sulfur dioxide concentrations were between 1, 200 ppm (3,400 mg/m^3) and 2,700 ppm, (7,700 mg/m^3) while sulfuric acid concentrations were in the range 55μg/m^3 to 174 μg/m^3. The smoke concentrations were presumably about 600 mg/m^3, although it is not clearly stated whether this was the concentration used in the combinations. The end point was the mean fatal dose. The administration, of smoke prior to exposure to the irritant substances did not alter the toxicity in contrast with a report by Pattle and Burgess [3] of a protective effect. The effect of smoke on the toxicity of the various irritants was highly variable when the two agents were given simultaneously. In guinea pigs the toxicity of sulfur dioxide was decreased by the smoke and in mice it was increased; the toxicity of sulfuric acid was increased by the smoke.

Gross *et al.*[5] reported the typical lesions of pneumontis in the lungs of hamsters, rats, and guinea pigs exposed to "a sufficiently large number of carbon particles with either adsorbed sulfur dioxide or nitrogen dioxide." Exposure was 8 hours per day, 5 days a week, for 4 weeks. The histological sections from animals killed about a month after the end of exposure, indicated that the lesions were persistent. The lesions were concentrated in the regions of the respiratory bronchioles and alveolar ducts and consisted of cellular wall thickening. Data were not provided about the amount of irritant absorbed by the activated carbon particles, or the particle size of the carbon, nor on the meaning of "a sufficiently large number of carbon particles." It was concluded that under certain experimental conditions the irritant gases were capable of rendering normally inert particles irritant, but these conditions were not defined.

2. Sulfur Dioxide and Sulfuric Acid

The effect of a combination of sulfur dioxide and sulfuric acid on guinea pigs has been reported.[6] Only one combination of concentrations was used, 89 ppm (255 mg/m^3) sulfur dioxide and 8 mg/m^3 sulfuric acid (1-μ MMD). Eight animals were exposed to this combination for 8 hours and 2 of these animals were reexposed 4 days later to the same combination. Since only one combination of concentrations was used in these experiments, they do not provide evidence for synergism, and the author concludes that further studies are needed before the results can be interpreted.

Bushtueva [7 8] reported that exposure of guinea pigs to 0.5 mg/m^3 sulfuric acid produced only slight lung irritation. When the sulfuric acid was combined with 0.3 ppm ($\sim$0.9 mg/m^3) sulfur dioxide, considerable changes in lung pathology were observed 2 months after a 2-week exposure. These changes included thickening of the interalveolar septa, formation of lymphatic folliculi, and perivascular and peribronchiolar fibrosis. Histamine content of the lungs increased concomitantly with the pathology. It is difficult to draw any unified conclusions of practical value from these experiments, since none was designed so that the joint toxic action could be satisfactorily assessed.

C. EVALUATION BY PULMONARY FUNCTION IN ANIMALS

The increase in pulmonary flow resistance has been used by Amdur *et al.*[9-11] to study the effect of simultaneous exposure of guinea pigs to irritant gas and to an aerosol. Three experiments deal with the situation in which both the gas and the aerosol are irritant. Two of these use sulfur dioxide and sulfuric acid mist,[10] and one uses sulfur dioxide and zinc ammonium sulfate.[11]

In the sulfur dioxide-sulfuric acid experiments the concentrations employed were high (about 100 ppm sulfur dioxide and 8 mg/m^3 to 15 mg/m^3 sulfuric acid). When the sulfuric acid had a particle size of $0.8\text{-}\mu$ MMD, the joint toxic action was synergistic. When the acid particles were larger, with an MMD of 2.5μ, the response to the mixture was slightly less than the response to the higher level of sulfur dioxide alone.

The concentrations used in the experiment with zinc ammonium sulfate and sulfur dioxide were at about the concentrations at which these substances were estimated to have been present during the Donora episode. Zinc ammonium sulfate, $0.29\text{-}\mu$ MMD, was used at 0.25 mg/m^3, and the sulfur dioxide was at 2 ppm (~ 6 mg/m^3). The effect of the combination demonstrated synergism as had been observed with the smaller-sized sulfuric acid. An explanation for these results may go beyond the simple hypothesis that adsorption of sulfur dioxide by the smaller-size acid particles, which penetrated the deeper areas of the lung, increased the amount of sulfur dioxide reaching the alveoli.

Other experiments have studied the combined effects of sulfur dioxide and "inert" aerosols (in the sense that they do not produce a statistically significant alteration in the flow-resistance of guinea pigs exposed for the standard 1-hour test period of this work).[10] The initial "inert" aerosol used as a standard was sodium chloride at a concentration of about 10 mg/m^3 in combination with a variety of irritant gases. This concentration of sodium chloride as an aerosol of $2.5\text{-}\mu$ MMD did not alter the response to sulfur dioxide. In all subsequent experiments a 1-percent sodium chloride solution was dispersed as an aerosol with a count mean size of about 0.04μ ($\sigma g = 3.3$). This aerosol did potentiate the response to the sulfur dioxide when tested over the concentration range 2 ppm to 200 ppm (~ 6 mg/m^3 to 570 mg/m^3).[10] The data were examined by comparing the concentration of gas plus aerosol with the concentration of gas alone required to produce the same percentage increase in resistance. Over the concentration range of 25 ppm to 250 ppm (72 mg/m^3 to 715 mg/m^3) a given concentration of sulfur dioxide plus aerosol produced about the same response as was produced by 2.5 to ? times that concentration of the gas alone.

At lower concentrations the relationship changed, and at 2 ppm the gas plus aerosol produced the same response as 30 times that concentration of gas alone. A comparison of the dose-response curves for sulfur dioxide and sulfuric acid (0.8μ) gave a similar picture.

At the higher concentration ranges, the response to sulfuric acid was uniformly 15 to 20 times the response to an equivalent concentration of sulfur dioxide, but at 0.5 ppm (1.4 mg/m^3) sulfuric acid the response is equivalent to the response to 60 ppm (170 mg/m^3) sulfur dioxide rather than to 8 ppm to 10 ppm (23 mg/m^3 to 29 mg/m^3) as would have been predicted. However, for the entire concentration range tested, response to sulfuric acid was about 3 times the response to an equivalent amount of sulfur dioxide in the presence of the aerosol, which suggested that the gas-aerosol mixture had the same biological effect as sulfuric acid. The implication is that the sulfur dioxide had become attached to the particles in some manner and created an irritant aerosol.

Decreasing the concentration of the sodium chloride aerosol to about 4 mg/m^3 decreased the potentiating effect.[12] The dose-response curve is parallel to that obtained with the higher concentration, but only 1 point, that at 20 ppm (57 mg/m^3), is greater than the corresponding point for sulfur dioxide alone. This decrease in potentiating effect with decreased aerosol concentration again suggested that the response was mediated through the formation of an irritant aerosol. It also suggested that the irritant aerosol formed by sodium chloride with sulfur dioxide was not a very potent irritant, since the effects had essentially vanished at an aerosol level of 4 mg/m^3.

Another factor suggesting the formation of an irritant aerosol comes from examination of the postexposure data. After exposures to gas-aerosol combinations were terminated, the resistance values remained above control values rather than returning to the preexposure level within an hour as had occurred after exposure to these levels of irritant gas alone.[13] As was pointed out at the end of Section D of Chapter 6, the return to control values was much slower when the irritant had been particulate matter than when it had been gaseous. The response to 113 ppm (323 mg/m^3) sulfur dioxide alone and to 26 ppm (74 mg/m^3) sulfur dioxide plus 10 mg/m^3 of NaCl aerosol was similar during the 1-hour exposure period. The animals exposed to the higher level of sulfur dioxide alone had shown a complete return to preexposure resistance values by 45 minutes after the exposure ended. Those exposed to the lower concentration plus aerosol still showed elevated resistance values 2 hours after the end of the exposure.

There is also evidence that the level of elevated resistance during the postexposure period is related to the concentration of the particulate material present. First of all, the time course of the response to various sulfur dioxide concentrations in the presence of a fixed sodium chloride aerosol concentration (10 mg/m^3) was examined. At the end of the exposure period the resistance values were higher at higher sulfur dioxide concentrations. One hour after the exposure ended they were all above control values but were all of the same order of magnitude, regardless of sulfur dioxide concentration. Two hours after the end of the exposure, the resistance values of the animals exposed to sulfur dioxide concentrations of 25 ppm (72 mg/m^3) or above had increased slightly. The animals exposed to 2 ppm ($\sim$6 mg/m^3) had returned to preexposure levels by 2 hours after the end of exposure.

Next, the total amount of aerosol was reduced in 2 ways: by reducing the concentration to 4 mg/m^3 for 1 hour, and by exposing animals for only half an hour to 10 mg/m^3.[9] The sulfur dioxide concentration was fixed at about 20 ppm ($\sim$60 mg/m^3). The results were entirely consistent with the hypothesis that there was a residual effect from an irritant aerosol which had been formed. An increase in resistance was observed between the first and second postexposure hours when the exposure had been for half an hour, and the resistance continued at about this level for the 5-hour postexposure observation period.

It has also been found that the potentiation of the response to sulfur dioxide by aerosols of sodium chloride is slow to develop.[9, 12] When the responses to gas alone, and to gas plus aerosol, are compared at 10 minutes there is no apparent difference. In this aspect the response is different from that produced by formaldehyde with sodium chloride [12] or that produced by aerosols of soluble metal salts known to catalyze the oxidation of sulfur dioxide to sulfuric acid.[9]

The solubility of sulfur dioxide in a liquid droplet appears to play some role in its potentiation by inert aerosols. This has been suggested [9] by the way in which the potentiating ability of aerosols of soduim chloride, potassium chloride, and ammonium thiocyanate is related to the solubility of sulfur dioxide in solutions of these salts. At the humidities prevailing in the exposure chamber, these substances would have been present as solid particles which, upon entering the moist respiratory tract, would take up water and become droplets.

Most solid aerosols tested [1] have not potentiated the response to sulfur dioxide, which suggests a role of solubility in potentiation. Thus, spectrographic carbon, activated coconut charcoal, iron oxide fume, triphenylphosphate, fly ash, and manganese dioxide at levels of 8 mg/m^3 or above, produced no detectable effect on the resistance nor did they potentiate the response to levels of sulfur dioxide ranging between 1 ppm and 100 ppm (3 ~mg/m^3 and ~290 mg/m^3). In several of the 19 groups of animals exposed to sulfur dioxide plus solid aerosols, the response was apparently less than that observed with a corresponding concentration of sulfur dioxide alone. The attenuations were not statistically significant for either group of animals exposed to what was termed only "fly ash from an oil-fired burner," one exposure being about 10 ppm (~30 mg/m^3) and another being at about 20 ppm (~60 mg/m^3) sulfur dioxide. When the response data from the two individual experiments were compared with those from response to 20 ppm sulfur dioxide alone, the attenuating effect was statistically significant. It is also mentioned that while triphenylphosphate aerosols of 0.3μ or 1μ failed to alter the response to sulfur dioxide even when present in concentrations of 50 mg/m^3, aerosols of 2μ had a striking attenuating effect when given with sulfur dioxide, and they also protected against the gas when given prior to exposure to sulfur dioxide.

The aerosols which appeared to have importance in the potentiation of sulfur dioxide were the aerosols of soluble salts which might catalyze the oxidation to sulfuric acid.[9] Aerosols of ferrous iron, manganese, or vanadium, when used at concentrations of 0.7 mg/m^3 to 1 mg/m^3 produced a potentiation of the response. A sulfur dioxide concentration of 0.2 ppm (~0.6 mg/m^3) produced a resistance increase of about 10 percent when given alone and an increase of about 35 percent when given in the presence of these aerosols. The potentiation was already observable at 10 minutes. It could be demonstrated by qualitative tests for sulfate that there was sulfate present on the aerosol collected from the chamber, which was not the case with sodium chloride aerosols.

These experiments suggest that the nature of the particulate material, as well as its size range are key factors. The aerosols of importance are those capable of dissolving sulfur dioxide and possibly of oxidizing it to sulfuric acid mist.

D. EVALUATION BY PULMONARY FUNCTION IN MAN

Frank *et al.*[11] examined the response of human subjects to levels of sulfur dioxide of about 1 ppm (~3 mg/m^3), 5 ppm (~15 mg/m^3), and 15 ppm (~43 mg/m^3), with and without the addition of sodium chloride aerosol. In the initial experiments, the agents were administered for 10-minute periods in sequence with a 15 to 20 minute recovery period between exposures. In a second series, the gas and gas plus aerosol exposures of each subject were made at least a month apart and were extended to 30 minutes. In the experiments in which the gas and the gas plus aerosol were given in sequence, the response to the second exposure was consistently lower than the response to the first exposure, no matter in which order the gas alone and gas plus aerosol were given. In the second series, when the exposures were a month or more apart, no difference was detected between the response to the gas alone and the response to the gas plus aerosol.

When the data for the control values and the resistance response for the same individual to the same level of sulfur dioxide on different occasions are examined, it is seen that a very dramatic potentiation would have been needed to yield positive results in this study. On one subject studied on seven occasions, the control resistance values ranged from 0.65 to 1.48 cm H_2O/l-sec, a difference of about 128 percent. The alterations produced by exposure on 3 different occasions to 15 ppm sulfur dioxide were 86 percent, 58 percent, and 156 percent increases in resistance. Two exposures to 15 ppm sulfur dioxide plus aerosol produced on one occasion an increase of 220 percent and on the other occasion an increase of 42 percent. The subject who showed the most extreme variation on three occasions had control resistance of 1.13 cm, and 1.46 cm H_2O/l-sec and corresponding resistance increases were 130 percent, 93 percent, and 410 percent during exposures to 15 ppm sulfur dioxide alone. The differences in response on the various occasions were not related to absolute level of control resistance, to functional residual capacity, to time of year, or to the previous total number of exposures. The variations on exposures to gas alone on different occasions were as great as the differences between the response to gas alone and to gas plus aerosol. The study makes apparent the difficulties of studies on humans.

The potentiating effects on guinea pigs are observed by establishing dose-response curves for a large sample of animals: such studies with humans are virtually impossible. The study of Frank *et al.*[14] does, however, present two important conclusions. The first is the demonstration of variability of response to sulfur dioxide on different occasions. The second relates to the data on control resistance values for the same individual on different occasions. The comparison of this variation with the magnitude of change produced by sulfur dioxide exposures serves to set the latter in proper physiological perspective.

Toyama[15] studied the flow resistance response of 13 subjects to sulfur dioxide with and without sodium chloride aerosol. Control measurements were made, and the subjects were then exposed in sequence for 5 minutes to sodium chloride, sulfur dioxide alone, and sulfur dioxide plus sodium chloride. The sulfur dioxide concentrations ranged from 1.6 ppm to 56 ppm (4.6 mg/m^3 to 160 mg/m^3). The sodium chloride aerosol produced no change in resistance. The sulfur dioxide produced an increase in resistance and the sulfur dioxide plus sodium chloride produced a greater increase than the gas alone in all subjects. Dose-response curves were plotted for the gas alone and for the gas plus aerosol. There was striking overall resemblance to the dose-response curves for the guinea pig experiments of Amdur.[10]

A comparison of equal response concentrations of gas alone and of gas plus aerosol shows similarity to the animal data in that the ratio is greater at the lower concentrations. The sodium chloride used in these studies had a CMD of 0.22μ. Nakamura[16] studied in a similar manner the effect of sodium chloride with a CMD of 0.95μ on the response of 10 subjects to sulfur dioxide. Again, in every case the response was greater when the sodium chloride aerosol was combined with the sulfur dioxide.

It is tempting to conclude that these studies[15 16] have demonstrated that the human subjects had behaved as did Amdur's guinea pigs. However, this conclusion is not necessarily valid. The exposures of human subjects were for 5 minutes, and it has been indicated[11 12] that in the guinea pig the potentiating effect of sodium chloride on sulfur dioxide is not apparent at 10 minutes. If the 2 species had been behaving in the same manner, a potentiation would not have been observed in these experiments.

When the exposures were given in sequence, the response to the second exposure (always the combination exposure) was invariably greater than the response to the first exposure. The results of sequential exposures to gas and to gas plus aerosol could be interpreted if data were given for a similar number of subjects exposed twice in this manner to the gas alone. In the 10-minute exposures reported by Frank *et al.*[14] the sequence of gas and of mixture was varied, but the response to the second exposure was less than the response to the first. As stated earlier, sequential exposures in experiments of this type are understandable as a practical procedure, but they constitute a "toxicological trap" for the unwary which unfortunately is not evaded merely by randomizing the sequence.

Burton *et al.*[17] found that the presence of 2 mg/m^3 to 2.7 mg/m^3 of sodium chloride aerosol below 1μ did not affect the response of 10 subjects to from 1 ppm to 3 ppm ($\sim$3mg/m^3 to $\sim$9mg/m^3) sulfur dioxide. There was no evidence of alteration of pulmonary mechanics immediately following exposures to this gas concentration, either with or without the added aerosol. This lack of response would have been predicted on the basis of animal experiments with 4 mg/m^3 sodium chloride.[12]

Toyama and Nakamura[18] examined the effect of aerosols of hydrogen peroxide with CMD values of 4.6μ and 1.8μ on the response to sulfur dioxde. They give CMD values of 4.6μ and 1.8μ for the aerosols, measured in a manner which does not necessarily relate to the airborne size. The hydrogen peroxide concentrations were of the order of 0.3 mg/m^3. Sulfur dioxide concentrations ranged from 1.5 ppm to 60 ppm (4.3 mg/m^3 to 170 mg/m^3). The sulfuric acid concentrations produced by the oxidation of sulfur dioxide were 0.8 mg/m^3 to 1.4 mg/m^3 for the larger aerosol and 0.01 mg/m^3 to 0.1 mg/m^3 for the smaller aerosol, but the reasons for the differences in acid concentration are not discussed.

The response to the mixture was greater than the response to sulfur dioxide alone. The response was greater with the larger aerosol than with the smaller aerosol and is in accord with the particle size effect discussed earlier. It is likely that it is also related to the concentration of sulfuric acid formed, since this was reported to be at least 10 times greater when the larger aerosol was used. When the sulfur dioxide exposures were grouped at 1 ppm to 10 ppm, 10 ppm to 30 ppm, and 30 ppm to 60 ppm, the response was graded with concentration in the presence of the smaller aerosol (lower acid concentration) but was uniformly about the same, an increase of 35 percent to 40 percent in resistance, for the 3 levels of gas when the larger particles (higher acid concentration) were present. These experiments present the same problem of sequential exposures discussed above and do not demonstrate synergism.

The data on the effect of particulate material on the response of human subjects does not consistently demonstrate a potentiation by sodium chloride aerosol or a synergism with sulfuric acid mist. There is some suggestion of such effects in three papers, but lack of adequate control data on repeated exposure to sulfur dioxide alone precludes proper interpretation of these data.

E. SUMMARY

The experiments reviewed use changes in pulmonary function, changes in pathology, and mortality to evaluate possible potentiating or synergistic effects of particulate matter on the toxicity of sulfur dioxide. Only changes in pulmonary function may be studied in experiments on man. Synergism by both irritant and "inert" particles is considered.

Sulfur dioxide in the atmosphere may be partly converted into the more irritant sulfuric acid, especially at high humidity and in the presence of particulate material. The irritant potency of sulfuric acid aerosol in itself is dependent on size and relative humidity.

The potentiation by particulate matter of toxic responses to sulfur dioxide (synergism) has been observed under conditions which would promote the conversion of sulfur dioxide to sulfuric acid. The degree of potentiation is related to the concentration of particulate matter. A threefold to fourfold potentiation of the irritant response to sulfur dioxide is observed in the presence of particulate matter capable of oxidizing sulfur dioxide to sulfuric acid. Aerosols of soluble salts of ferrous iron, manganese, and vanadium have been observed to produce this potentiation, although the concentrations used (0.7 mg/m^3 to 1.0 mg/m^3) were considerably greater than any reported levels of the metals in urban air.

Experiments with normal human subjects have failed to demonstrate any consistent potentiation of response to sulfur dioxide by sodium chloride particles. In the guinea pig, sodium chloride is the least effective of the various soluble aerosols that produce any potentiation.

F. REFERENCES

1. Schnurer, L. "Effects of Inhalation of Smoke from Common Fuels." Am. J. Pub. Health, Vol. 27, pp. 1010–1022, 1937.
2. Vintinner, F. J. and Baetjer, A. M. "Effect of Bituminous Coal Dust and Smoke on the Lungs—Animal Experiments. I. Effects on Susceptibility to Pneumonia." Ind. Hyg. & Occup. Med., Vol. 4, pp. 206–216, 1951.
3. Pattle, R. E. and Burgess, F. "Toxic Effects of Mixtures of Sulfur Dioxide and Smoke with Air." J. Pathol. Bacteriol., Vol. 73, pp. 411–419, April 1957.
4. Salem, H. and Cullumbine, H. "Kerosene Smoke and Atmospheric Pollutants." Arch. Environ. Health, Vol. 2, pp. 641–647, June 1961.
5. Gross, P., Rinehart, W. E., and deTreville, R. T. "The Pulmonary Reactions to Toxic Gases." Am. Indust. Hyg. Assoc. J., Vol. 27, pp. 315–321, 1967.
6. Amdur, M. O. "Effect of a Combination of SO_2 and H_2SO_4 on Guinea Pigs." Pub. Health Reports, Vol. 69, pp. 503–506, May 1954.
7. Bushtueva, K. A. "Toxicity of H_2SO_4 Aerosol." Gig. i Sanit., Vol. 22, pp 17–22, 1957. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. A Survey, Vol. 1,

Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., Jan. 1960, pp. 63–66.

8. Bushtueva, K. A. "Experimental Studies on the Effect of Low Oxides of Sulfur Concentrations on the Animal Organism." In: Limits of Allowable Concentrations of Atmospheric Pollutants, Book 5, Translated by B. S. Levine, U.S. Dept. of Commerce, Office of Technical Services, Washington, D.C., March 1962, pp. 92–102.
9. Amdur, M. O. and Underhill, D. "The Effect of Various Aerosols on the Response of Guinea Pigs to Sulfur Dioxide." Arch. Environ. Health, Vol. 16, pp. 460–468, 1968.
10. Amdur, M. O. "The influence of Aerosols Upon the Respiratory Response of Guinea Pigs to Sulfur Dioxide." Am. Ind. Hyg. Assoc. Quart., Vol. 18, pp. 149–155, June 1957.
11. Amdur, M. O. and Corn, M. "The Irritant Potency of Zinc Ammonium Sulfate of Different Particle Sizes." Am. Ind. Hyg. Assoc. J., Vol. 24, pp. 326–333, July–Aug. 1963.
12. Amdur, M. O. "The Effect of Aerosols on the Response to Irritant Gases." In: Inhaled Particles and Vapors, C. N. Davies (ed.), Proc. Intern. Symposium, Oxford, March 29–April 1, 1960, Pergamon Press, Oxford, 1961, pp. 281–292.
13. Amdur, M. O. "The Physiological Response of Guinea Pigs to Atmospheric Pollutants." Intern. J. Air Pollution, Vol. 1, pp. 170–183, Jan. 1959.
14. Frank, N. R., Amdur, M. O., and Wittenberger, J. L. "A Comparison of the Acute Effects of SO_2 Administered Alone or in Combination with NaCl Particles on the Respiratory Mechanics of Healthy Adults." Intern. J. Air Water Pollution, Vol. 8, pp. 125–133, 1964.
15. Toyama, T. "Studies on Aerosol. I. Synergistic Response of the Pulmonary Airway Resistance on Inhaling Sodium Chloride Aerosols and SO_2 in Man." Japan J. Ind. Health, Vol. 4, pp. 86–92, 1962.
16. Nakamura, K. "Response of Pulmonary Airway Resistance by Interaction of Aerosols and Gases of Different Physical and Chemical Nature." Japan J. Hyg., Vol. 19, pp. 322–333, Dec. 1964.
17. Burton, G. G., Corn, M., Gee, J. B. L., Vassallo, C., and Thomas, A. "Absence of 'Synergistic Response' to Inhaled Low Concentration Gas-Aerosol Mixtures in Healthy Adult Males." (Presented at 9th Annual Air Pollution Medical Research Conference.) Arch. Environ. Health, Vol. 18, pp. 681–692, 1969.
18. Toyama, T. and Nakamura, K. "Synergistic Response of Hydrogen Peroxide Aerosols and Sulfur Dioxide to Pulmonary Airway Resistance." Ind. Health, Vol. 2, pp. 34–45, March 1964.

Chapter 9

EPIDEMIOLOGICAL APPRAISAL OF SULFUR OXIDES

Table of Contents

Page

A. INTRODUCTION 117

B. APPLICATION OF EPIDEMIOLOGY TO AIR POLLUTION STUDIES 117
  1. Indices 117
  2. Cautions 118

C. INDICES OF HUMAN RESPONSE: THE EPIDEMIOLOGIC STUDIES 119
  1. Acute Episodes 119
    a. Mortality 119
    b. Morbidity 125
  2. Chronic (Long-Term) Air Pollution 126
    a. Day-to-Day Variations in Mortality and Morbidity 126
    b. Geographical Variations in Mortality 127
      1. Studies Based on Available Data 127
      2. Special Studies Involving the Collection of New Data 128
    c. Geographical Variations in Morbidity—Special Studies 130
    d. Morbidity—Incapacity for Work 135
  3. Studies of Children 137
  4. Studies of Pulmonary Function 139
  5. Studies of Panels of Bronchitic Patients 140
  6. Miscellaneous Studies 142

D. EFFECTS OF INDUSTRIAL EXPOSURE 142

E. SUMMARY 144

F. REFERENCES 147

List of Figures

Figure

9–1 Mortality Figures for the January 1956 and December 1957 Smog "Episodes" in London. 121

9–2 Death Rate and Air Pollution Levels in Dublin, Ireland, for 1938-1949. 124

9–3 Age-Standardized Morbidity Rate per 1,000 for Three Diseases in Japan. 132

9–4 Incidence of Respiratory Disease Lasting More Than Seven Days in Women Versus Concentration of Sulfates in the City Air at Test Sites. 135

Page

9–5 Effect on Bronchitic Patients of High Pollution Levels (January 1954). 140

9–6 Comparison of Person-Days of Acute Illness with Seven Levels of Sulfur Dioxide Exposure in Chicago for Patients With Severe Chronic Bronchitis (Age 55 or More) for October-November 1967. 141

List of Tables

Table

9–1 Survey of Selected Acute Air Pollution Episodes in Greater London. 125

9–2 Pollution Levels in Salford. 129

9–3 Industrial Exposures. 144

BLANK PAGE

Chapter 9

EPIDEMIOLOGICAL APPRAISAL OF SULFUR OXIDES

A. INTRODUCTION

Health effects produced by atmospheric sulfur oxides are discussed in this chapter in terms of epidemiologic studies. Because sulfur oxides tend to occur in the same kinds of polluted atmosphere as particulate matter, few epidemiologic studies have been able adequately to differentiate the effects of the two pollutants. It follows, therefore, that the studies presented in this chapter are frequently identical with those described in the companion document, *Air Quality Criteria for Particulate Matter.*

Epidemiologic studies, as distinguished from toxicologic or experimental studies, analyze the effects of pollution from ambient exposure on groups of people living in a community. Such studies have the advantage of examining illness where it occurs naturally, rather than in a laboratory, but carry the disadvantage of not being able to control precisely all the factors of possible importance. Nevertheless, the preparation of air quality criteria must rest on epidemiologic studies because of the very severe limitations of toxicologic and industrial studies for this purpose. Other countries, notably the Netherlands and Sweden, have based their air quality criteria solely on epidemiologic studies.

In determining whether or not an association is causal, consideration must be given to several aspects of association which include strength, consistency, specificity, temporality, biological gradient, plausibility, coherence, and analogy.[1] A judgment of the value of an epidemiologic study requires an understanding of these aspects.

Many types of epidemiologic evidence suggest that air pollution may exert considerable influence on the health, as well as on the "satisfaction with life," of major segments of the world population.

Several health indices are described in Section B–1; certain precautions which should be observed in the application of epidemiologic methods to air quality criteria are suggested in Section B–2. The studies themselves are listed in Section C, according to the index employed. Several industrial exposures to the oxides of sulfur are mentioned in Section D, discussing their relevance to the epidemiologic studies.

B. APPLICATION OF EPIDEMIOLOGY TO AIR POLLUTION STUDIES

1. Indices

Various indices of health may be used for correlation with air pollution by the oxides of sulfur. Among the possible indices are:

1. Mortality (greater than expected):
   - (i) Deaths from all causes
   - (ii) Deaths from specific causes
   - (iii) Deaths among the different age and sex groups
2. Morbidity:
   - (i) Incidence of disease — chronic bronchitis, pulmonary emphysema, diffuse interstitial pneumonitis, cancer of respiratory tract, disease of remote organ (e.g., gastro-intestinal, ophthalmic, and cardiovascular systems)
   - (ii) Prevalence of diseases—same examples as for "incidence"
   - (iii) Prevalence of respiratory symptoms (e.g., changes in quality and/or quantity of sputum production)

(iv) Exacerbation of diseases—rhinorrhea, asthma, tracheobronchitis, and chronic illness and enhancement of infection: pneumonia, sinusitis, otitis, mastoiditis

(v) Changes in clinical conditions (e.g., bronchitic patients)

3. Changes in various aspects of lung function:

   (i) Ventilatory function—decrease in peak flow rate, decrease in spirometric volumes, impairment of flow-volume relationships, and increased airways resistance

   (ii) Blood/gas distribution—impairment of lung-gas distribution

   (iii) Blood/gas exchange — impairment of pulmonary blood-gas exchange

   (iv) Increased work of breathing

Definitions of the various disease states are to be found in the glossary; most of the pulmonary function methods have been mentioned in Chapters 9 and 10 of the companion document, *Air Quality Criteria for Particulate Matter.*

The manner of presentation of the state of epidemiological knowledge of effects of sulfur dioxide in the ambient atmosphere when accompanied by particulate matter is outlined in the Table of Contents.

2. Cautions

In the first place, as discussed in Chapter 2, methods of measurement vary from country to country and place to place. Results from the various methods of measurement are frequently very dissimilar.

Secondly, pollution and health indices are not always measured over the same time periods. It is to be hoped that the pollution levels cited bear some relation to those extant during the time when the chronic disease states were developing. Further, acute effects require frequent short-term pollution measurements to enhance detection, while long-term chronic processes may be adequately related to long-term sampling intervals. Air pollution measurements useful in studies of acute health effects are becoming available; a less satisfactory situation exists for the long-term effects studied.

Thirdly, in many instances the possible role of cigarette smoking has not been considered. It is expected that future epidemiologic studies involving adults will routinely collect data on smoking habits of the study group. Other factors are significantly related to respiratory disease. These include occupational and other past exposures; infections, past and present; and allergy and heredity.

Few or no epidemiologic studies have been possible where the pollution challenge has been *limited* to oxides of sulfur, unaccompanied by *significant* amounts of other pollutant substances. Indeed, most of the available conclusions link sulfur dioxide levels with those of concurrently measured particulate matter; some studies attempt statistical separation of the culpability of one factor from the other in the effects cited.

In seeking the possible effects on populations resident in areas of differing air pollution, factors such as smoking, type and conditions of employment, ethnic group, and mobility in response to experienced irritation or disease have sometimes been considered. There has, however, been a minimum of attention paid to the indoor or domestic environments and their potential contribution. Measurement of such indoor exposures might be difficult, but omission of the information could well modify the appraisal of the importance of pollution by the oxides of sulfur.

Toxicologic studies indicate a specific potential of some oxides of sulfur to produce human responses. The levels used in toxicologic studies are far higher than those found in the communities in the epidemiologic studies under review. Thus the actual responsibility of oxides of sulfur for the community responses is uncertain, and it is sometimes necessary to invoke additional concepts; for example, the idea of synergism with other known or unknown ambient pollutants, or the idea that sulfur dioxide is but an index of availability of some other substance(s) which are fully responsible for the effects reported.

Over a short period of time, mortality fluctuates considerably, and only a systematic, long-term approach will allow a valid determination of the real role of air pollution. Cassell *et al.*[2] have reviewed the problem of detecting peaks in mortality and relating them to any single variable. The danger with episodic studies is that short-term fluctuation in the death rate, when picked to coincide with an air pollution incident, may appear to be causally related; in the long term, however, numerous other unassociated peaks are found in both the death rates and the air pollution levels.

The concept of "susceptible population" demands consideration. Human responses to toxicants, and to community air pollution, show wide variations, which contribute in no small way to the difficulty in assessing in a general manner the effects of pollutants. Since air quality criteria must, unless otherwise specified, consider "all" population rather than just major segments of it, studies must consider especially the impact of air pollution on the "most sensitive" responders. Many factors seem to enhance susceptibility or sensitivity to air pollution. These include being at the extremes of age, (i.e., infants and the very old); having pre-existing chronic respiratory disease (e.g., pulmonary emphysema or chronic bronchitis); having preexisting cardiovascular disease (functional capacity not defined); regularly smoking cigarettes; or living in overcrowded or depressed socioeconomic strata. Some of these factors have been singled out for attention in the references to be cited, and the level of pollutant said to have an "effect" may take cognizance of such special sensitivity.

The effects discussed are related insofar as possible to specific pollution over specific time intervals; it must be emphasized that lower values by no means imply a "no effect" level of the pollutants.

C. INDICES OF HUMAN RESPONSE: THE EPIDEMIOLOGIC STUDIES

1. Acute Episodes

a. Mortality

The first well studied air pollution episode occurred between December 1 and 5, 1930, in the Meuse Valley, Belgium, when a heavy fog covered the entire valley. Several hundred people were severely ill with respiratory symptoms, and 63 died. Although no precise measurements were made during the episode, Firket estimated that a number of pollutants rose to high levels and that sulfur dioxide and sulfuric acid, which may have reached a total of 25,000 $\mu g/m^3$ or higher ($\sim$9 ppm), were probably the chief cause of the illness.[3, 4]

During late October 1948, Donora, Pennsylvania was blanketed by a dense fog, and 43 percent of the population was affected to some degree. Twenty persons died during or shortly after the smog, and 10 percent of the population was classified as severely affected. No measurements were taken during the episode, but a detailed study afterwards[4] concluded that no single agent was responsible, and that the observed effects were due to a chemical agent together with particulate matter. Sulfur dioxide and its oxidation products were undoubtedly significant contaminants. During subsequent inversion periods, presumably not as severe as the one in October 1948, daily averages of sulfur dioxide as high as 0.4 ppm ($\sim$1140 $\mu g/m^3$) were recorded.[5]

In writing about the Meuse Valley episode in 1936, Firket stated that if there were a similar phenomenon in London, some 3200 deaths might occur. Unfortunately he was quite accurate in his estimate since, in December 1952, the world's most disastrous smog incident occurred in London, causing about 4000 excess deaths throughout the Greater London area. Marked increases were noted in both respiratory and cardiovascular deaths (and for almost every cause except traffic accidents; presumably the smog was too thick for people to drive). Since some of the diseases such as lung cancer and tuberculosis were obviously existent before the pollution episode, much of the effect of the fog was clearly to hasten the death of people who were already ill. Detailed investigations were made of 1,280 post-mortem reports of persons who had died before, during, or shortly after the episode. No fatalities were found which could not have been explained by previous respiratory or cardiovascular lesions. In this episode as in others, the elderly and persons with pre-existing pulmonary and cardiac disease were most susceptible.

The maximum daily concentration of sulfur dioxide recorded during the 1952 London smog was 1.34 ppm (about 4,000 $\mu g/m^3$)[*] which appeared on the third and fourth day of the fog. Corresponding smoke levels were 4.46 mg/m^3 ($\sim$4500 $\mu g/m^3$). These figures are from County Hall, where only volumetric (hydrogen peroxide) measurements of SO_2 were made.[6]

Greater London has had several air pollution episodes before and since the large one of December 1952, but none has come close to causing 4,000 excess deaths.

A number of investigations have analyzed and compared the various London episodes. The report by Brasser *et al.*[7] appears to cover all the episodes and to present a relatively detailed analysis of each of these episodes, pointing out the importance of the duration of the maximum values. More recently, Joosting[8] has examined the relationship between the duration of maximum values of sulfur dioxide and smoke during air pollution episodes, as well as the differential relationship between sulfur dioxide and smoke levels and the resulting mortality.

In conurbations, such as London, New York, Chicago, and Detroit, it has been possible to observe deviations from the moving averages of deaths during various seasons, and to relate such deviations to the coincident period levels of air pollutants.[9]

Gore and Shaddick[10] and Burgess and Shaddick[11] reviewed acute "fog" episodes which occurred in London in 1954, 1955, 1956 (January and December), and 1957 (December), in terms of excess mortality above a moving average, related to the mean of daily readings at seven stations for smoke and SO_2. Figure 9–1 shows mortality figures for the January 1956 and December 1957 episodes. Somewhat differing patterns of onset of mortality rise, of age of population suffering most heavily, of deaths related to bronchitis and to other respiratory diseases, and of total deaths, were noted in these acute episodes. Common to them, however, were elevations of mean daily levels of SO_2 and smoke measured at seven different stations from two to four times the winter average levels; "effects" were estimated at 2,000 $\mu g/m^3$ black suspended matter together with 1145 $\mu g/m^3$ (0.4 ppm)[*] of sulfur dioxide (representing all acidic gases). Deaths ascribed to bronchitis were materially affected, but deaths due to other causes also increased. Deaths appeared to begin to increase before the onset of the episodes; during the episodes, of course, they increased substantially. Scott[12] observed a similar relationship, for similar periods of "fog," with "effective pollutant" levels at seven different stations in London of 2,000 $\mu g/m^3$ for smoke and 0.4 ppm for SO_2 (reported by Scott as 1,140 $\mu g/m^3$).[**] There was a sharp impact on the elderly and the greatest proportionate rise, for cause of death, in bronchitics.

Martin and Bradley[13] correlated daily mortality (all causes) and daily bronchitis mortality with mean daily black suspended matter for the winter of 1958–1959, and also found a significant positive association between mean daily sulfur dioxide levels and deaths (all causes). Bronchitis deaths showed a lower correlation with the pollution level, and the authors suggest the need for consideration of effects of air pollution on patients with cardiovascular disease. In additon, excess deaths have been related to increases (on the day preceding death) of mean daily black suspended matter by more than 200 $\mu g/m^3$, and rises in mean daily SO_2 of more than about 75 $\mu g/m^3$ (2.5 parts per hundred million).[*] In a later paper[14], data are shown to suggest an increase in mortality from all causes, and of respiratory and cardiac morbidity, associated with levels of smoke about 1,000 $\mu g/m^3$, and SO_2 concentrations of 715 $\mu g/m^3$ (25 pphm). This "effect" is properly related to abrupt rises in the concentrations of smoke and/or SO_2, with perhaps a continuum of effects at lower levels. Since these measurements were obtained at a single point in Central London, it should be presumed that a relatively wide range of values around these levels actually contributed to the mortality statistics which were correlated. A re-analysis by Lawther[15] of these mortality studies places the mortality "effect" at about 750 $\mu g/m^3$ for smoke and 715 $\mu g/m^3$ (0.25 ppm) for SO_2. Joosting[8] states that the maximum sulfur dioxide concentration above which significant correlations occur with death and disease is 400 $\mu g/m^3$ to 500 $\mu g/m^3$ (0.15 ppm to 0.19

---

[*] Unless otherwise stated, British measurements of SO_2 concentrations are obtianed by the hydrogen peroxide titrimetric method and may be higher than the actual SO_2 concentration due to the presence of other acidic gases (see Chapter 2).

[*] SO_2 values in this study were originally reported in ppm.

[**] SO_2 is converted from ppm to $\mu g/m^3$ in Scott's report by using an equivalency 2,850 $\mu g/m^3$=1 ppm. This report uses 2,860 $\mu g/m^3$=1 ppm, and for consistency attempts to express the value (0°C, 760 mm Hg) in $\mu g/m^3$ first. Most of the American SO_2 values are measured at 25°C, 760 mm Hg; the equivalency under these circumstances is 2,610 $\mu g/m^3$=1 ppm.

[*] SO_2 values in this study were originally reported in parts per billion (ppb).

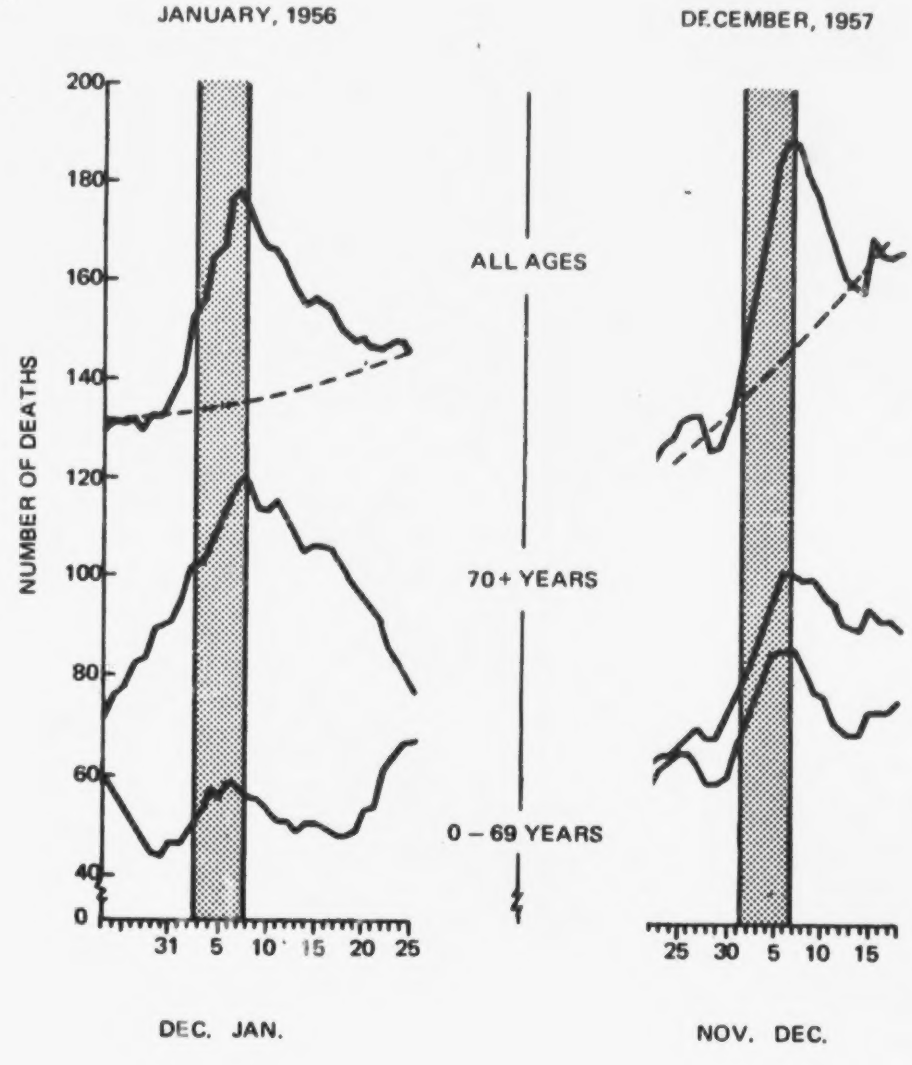

Figure 9-1. Mortality Figures for the January 1956 and December 1957 Smog "Episodes" in London.[10]

The figure shows the increase in numbers of deaths during smog "episodes" (shaded periods), especially in the older age group.

ppm)** when there is a high soot content.

The Dutch report on sulfur dioxide,[7] which discusses in detail seven air pollution episodes in London, states that in the December 1956 episode, 400 excess deaths, or 25 percent above expected, were observed in Greater London at maximum 24-hour levels of 1,200 $\mu g/m^3$ for smoke and 1,100 $\mu g/m^3$ for SO_2 (0.4 ppm).** The report also notes that in January 1959, 200 excess deaths were observed in Greater London, or 10 percent above the expected mortality, at a level of 1,200 $\mu g/m^3$ for smoke and 800 $\mu g/m^3$ (0.30 ppm)** for SO_2.[7] The episodes all took place during winter; cold weather seems to be an important factor in London air pollution mortality.

In Martin's review [11] in 1964 of daily mortality in London during the winters of 1958–1959 and 1959–1960, he concluded: "From the data it would be difficult to fix any threshold value below which levels of air pollution might be regarded as safe." However, his review included data with sulfur dioxide concentrations ranging upward from about 400 $\mu g/m^3$ (0.14 ppm), and accompanied by smoke concentrations of 500 $\mu g/m^3$ and above.

As a result of smoke control regulations, the particle content of London air has steadily decreased since the 1950's but the sulfur dioxide concentrations have not decreased proportionately. At the same measuring sites as in 1952, sulfur dioxide was actually slightly higher in the 1962 episode than in that of 1952, but smoke levels were considerably lower. Also, as Brasser *et al.* have noted, there was only one day of maximum pollution values in 1962 as contrasted with the two days of maximum pollution in December 1952.[7] Since 1952, a great deal of publicity has been given to the harmful effects of smog, and more susceptible individuals have been encouraged to use masks and filters and stay indoors. In addition, when episodes come close together, a large number of susceptible individuals might not accumulate, since some are killed off each time. An effect as large as that seen in the first incident would not, therefore, be expected.

The number of deaths in New York City was reviewed for excess mortality in relation to the air pollution episode of November 1953 by Greenburg *et al.*[16] Excess deaths were related to elevations of concentrations of sulfur dioxide and suspended particles. Average daily smoke shade measured in Central Park was in excess of 5.0 coh units, while the SO_2 rose from the New York City average ranges of 430 $\mu g/m^3$ to 570 $\mu g/m^3$ (0.15 to 0.20 ppm)* to a maximum level of 2,460 $\mu g/m^3$ (0.86 ppm), probably a half-hour value. For this episode, there was a "lag effect," and distribution of excess deaths among all age groups was noted. The number of deaths, although not showing the marked rise seen in some of the London episodes, was above average for comparable periods in other years during and immediately after the incident. For the November 15 to 24, 1953, period, the average number of deaths per day was 244, whereas during the 3 years preceding and following 1953, the average was 224 deaths per day for the same calendar period.

A later episode (1962) was studied, but Greenburg *et al.*[17] did not discern an excess mortality. However, McCarroll and Bradley,[18] reviewing episodes in New York City in November and December of 1962, January and February of 1963, and February and March of 1964, compared 24-hour average levels of various pollutants with New York City mortality figures, employing daily deviations from 15-day moving average; the measurements were performed at a single station in lower Manhattan, and fluctuations in the values at this station were known to correlate well with those at another station 6.5 miles away. Excess deaths on December 1, 1962, followed a daily average sulfur dioxide concentration of 2,600 $\mu g/m^3$ (0.72 ppm)* and smoke shade in excess of 6 coh units, during a period of atmospheric inversion and low ground-wind speed. The increased death rates were shared by the 45

** SO_2 is converted from ppm to $\mu g/m^3$ in the Dutch reports by using the equivalency, 2700 $\mu g/m^3$=1 ppm. This report uses 2,860 $\mu g/m^3$=1 ppm whenever possible.

* SO_2 values in this study were originally reported in ppm.

to 64 age group, as well as by the age group over 65. In a later episode, January 7, 1963, associated with an SO_2 concentration of about 1,715 $\mu g/m^3$ (0.6 ppm)* and a smoke shade value of 6 coh units, there was a peak death rate apparently superimposed upon an elevated death rate average due to the presence of influenza virus in the community.

Another severe episode of air pollution encompassed the New York City area during the Thanksgiving weekend, November 23 to 25, 1966. The maximum 24-hour average of hourly SO_2 values was 1,460 $\mu g/m^3$ (0.51 ppm)* (electroconductivity) on November 23, and 1,344 and 1,175 $\mu g/m^3$ (0.47 and 0.41 ppm) on the 24th and 25th. The maximum hourly concentration was 2,915 $\mu g/m^3$ (1.02 ppm). Smoke shade values were above 5 cohs on the 3 days. The average number of daily deaths during the 7 days of the air pollution episode was 261 compared to the expected value of 237 for control periods in 6 surrounding years.[19]

An example of the inherent danger of relating mortality peaks to air pollution is shown by Leonard *et al.*[20] in the Dublin studies. During the war and post-war years of 1941 to 1947, peat was burned as the main fuel rather than coal, and air pollution (as measured by particle concentrations) was markedly decreased. Sulfur dioxide levels varied in a manner similar to those of suspended particulate matter. The winter peaks in death, however, were unaffected, and thus do not seem to be related to air pollution. When coal again became available in 1948, the air pollution levels rose with no apparent effect on the death rate (see Figure 9–2). Unfortunately, it is not possible to assess the effect of changing medical practices and the advent of antibiotics for use in treating respiratory diseases on these data.

In Detroit[21] a rise in infant mortality and deaths in cancer patients occurred over a 3-day period accompanied by a rise in the 3-day mean suspended particulate matter for the same period above 200 $\mu g/m^3$ and accompanied by an instantaneous SO_2 maximum of 2,860 $\mu g/m^3$ (1.0 ppm)* (September 1952). This is not believed to be related to the cold temperatures which have characterized the London episodes.

In Osaka, Japan, Watanabe[22] reported on excess deaths in a December 1962 smog episode. There were 60 excess deaths related to mean daily concentrations of suspended matter greater than 1,000 $\mu g/m^3$, with accompanying sulfur dioxide greater than 285 $\mu g/m^3$ (0.1 ppm)*; the measurements were made at a single station in the central commercial area of the city.

In an effort to include all the available relevant data, we must mention the discussion by Brasser *et al.*[7], calling attention to the availability of data for Rotterdam. He says:

> "From investigations at Rotterdam indications have been obtained that there exists a positive association with the total mortality if the value of 500 $\mu g/m^3$ [0.19 ppm]* per 24 hours is surpassed for a few days. Perhaps this effect begins to be active at lower concentrations already present. There is a faint indication that this will happen somewhere between 300 and 500 μg SO_2 per m^3 [0.11 to 0.19]* per 24 hours."

The Rotterdam episodes of January–February 1959 and December 1962 have also been discussed by Joosting.[8] What is especially significant is that particulate levels are generally low in Rotterdam. On comparing particulate and SO_2 concentrations, Joosting has characterized the ratio of particulates to SO_2 as 1:1, 1:1.5 to 1:2, and 1:3 to 1:4. Rotterdam is in the last category, whereas London is in the first. Measurements in Rotterdam are made with the hydrogen peroxide titrimetric method.

When a marked increase in air pollution is associated with a sudden dramatic rise in the death rate or illness rate lasting for a few days and both return to normal shortly thereafter, a causal relationship is strongly suggested. Sudden changes in weather, however, which may have caused the air pollution incident, must be considered as another possible cause of the death rate increase. Over the years, a number of such acute epi-

---

* SO_2 values in this study were originally reported in ppm.

* The numbers in brackets are the editor's; an equivalency of 2,700 $\mu g/m^3$=1 ppm SO_2 was used to assure consistency with any conversions made by the authors.

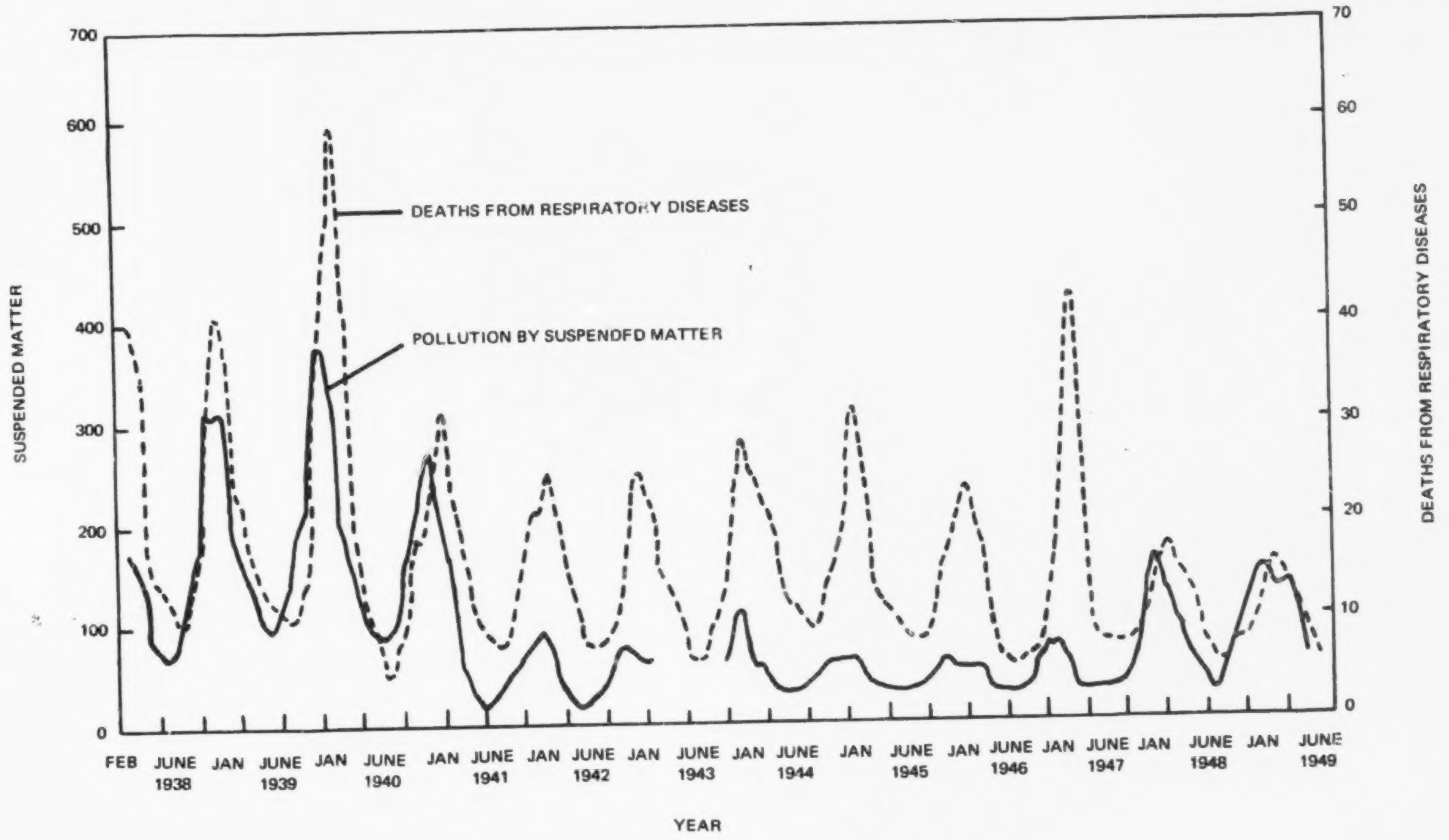

Figure 9-2. Death Rates and Air Pollution Levels in Dublin, Ireland for 1938-1949.

sodes have been reported, and there seems little doubt that air pollution was the cause.

Table 9–1 is an attempt by Brasser *et al.*[7] to summarize some of the recent major air pollution episodes in Greater London.

The British studies presented in this section suggest that excess mortality, a small rise in the daily death rate, is detectable in large populations if the concentrations of sulfur dioxide rise abruptly to levels at or about 715 $\mu g/m^3$ (about 0.25 ppm) in the presence of smoke at 750 $\mu g/m^3$. The major targets are the aged population, patients with chronic obstructive pulmonary disease, and patients with cardiac disease. A more distinct rise in deaths is noted generally when sulfur dioxide exceeds 1,000 $\mu g/m^3$ (about 0.35 ppm) for 1 day and particulate matter reaches about 1,200 $\mu g/m^3$. Daily concentrations of sulfur dioxide in excess of 1,500 $\mu g/m^3$ for 1 day ($\sim$0.5 ppm) in conjunction with levels of suspended particles exceeding 2,000 $\mu g/m^3$ appear to be associated with an increase in the death rate of 20 percent or more over base line levels. This same effect is observed at lower sulfur dioxide levels if the maximum pollution levels last for a longer period.

b. Morbidity

The acute episodes have resulted in substantial increases in illness. Thus a survey[23] of emergency clinics at major New York City hospitals in November 1953 indicated a rise in visits for upper respiratory infections and cardiac diseases in both children and adults in all of the four hospitals studied. Sulfur dioxide ranged between 200 $\mu g/m^3$ and 2,460 $\mu g/m^3$ (0.07 ppm to 0.86 ppm)* during the period from November 12 to 24, and hospital admissions were clearly elevated by November 16, at which time concentrations had not yet exceeded 715 $\mu g/m^3$ (0.25 ppm); "smoke shade" at this time was close to 3 coh units.

Again, the number of emergency clinic visits for bronchitis and asthma at seven large New York City hospitals was examined during the Thanksgiving 1966 air pollution episode.[24] There was a rise in the number of such visits on the third day of the episode, among the patients age 45 and over, at three of the seven hospitals investigated. Unfortunately, the Thanksgiving holiday greatly

---

* SO_2 values in this study were originally reported in ppm.

Table 9–1.—SURVEY OF SELECTED ACUTE AIR POLLUTION EPISODES IN GREATER LONDON.[7]

	Dec. 1952	Jan. 1956	Dec. 1962	Dec. 1957	Dec. 1956	Jan. 1955	Jan. 1959
Duration of the cumulation period in days	5	5	5	5	10	11	5
Number of days with maximum pollution	2	2	1	-	5	1x3[a]	1
SO_2 level preceding episode	500	300	400	300	300	300	300
SO_2 maximum	4000	1500	3300	1600	1100	1200	800
SO_2 increase per day	1200	500	1000	325	400	450	250
Soot level preceding episode	400	500	200	400	400	500	400
Soot maximum	4000	3250	2000	2300	1200	1750	1200
Soot increase per day	1200	1300	600	500	400	600	400
Number of excess deaths	3900	1000	850	800	400	240	200
Number of days with excess mortality	18	10	13	10	6	6	6
Daily mortality expected under normal circumstances	300	330	310	300	270	320	325
Average daily mortality in the period (excess mortality as a percent of normal)	170	130	120	125	125	112	110

Remark: The SO_2 and soot concentrations mentioned are average values over 24 hours expressed in $\mu g/m^3$.

[a] Maximum pollution values of one day's duration occurred three times.

complicated evaluation of the emergency clinic visits over the holiday period.

In the investigation of the London episode of December 1952, information on illness was collected from as many sources as possible including sickness claims, applications for hospital admission, pneumonia notifications, and records of physicians. The analysis demonstrated a real and important increase in morbidity, though there was some indication that the increase in illness was not as large proportionately as the increase in deaths and the effects were not so sudden in producing a marked rise in the early days of the episode. In a number of other severe London episodes the increase in morbidity put a considerable strain on the health services.

These episodes reflect results which fall into Level IV of the World Health Organization's "guides to air quality."[25] These "guides," equivalent in usage to our term "criteria," cover sets of concentrations and exposure times at which specified types of effects are noted or at which no effect is noted. Level IV includes "concentrations and exposure times at and above which there is likely to be acute illness or death in susceptible groups of the population."

2. Chronic (Long-Term) Air Pollution

Kurland[26] has called to our attention the fact that the air pollution episodes represent, by definition, episodes of massive, overwhelming, and unusual exposure, and thus the most significant pathologic effects. There is an "iceberg" effect in that such data represent the obvious, while the greater share of the problem remains submerged. We are dealing with an essential dose-response situation, the upper limits of which are represented by these episodes.

a. Day-to-Day Variations in Mortality and Morbidity

In a systematic approach to analyzing respiratory and cardiac morbidity daily in London, Martin[14] examined deviations in morbidity from a 15-day moving average in London during the winters of 1958–1959 and 1959–1960. Both smoke and sulfur dioxide concentrations appear to be about equally related to morbidity rates, and a definite excess in morbidity seemed to exist as it did for mortality, though there was a somewhat greater degree of irregularity.

An approach similar to Martin's, but limited to observations on mortality, was used by McCarroll and Bradley[18] in New York City. Covering a 3-year period (1962–1964) they examined a number of peaks in New York City mortality associated with periods of high air pollution. There are examples given where sulfur dioxide and smoke shade appear to be related to mortality. The authors present other episodes, however, where the relationship to air pollution is not nearly as clear, although the death rate fluctuates to even higher peaks. Reference to this analysis has already been made in our discussion of the data on episodes in Section B–1.

The rate of hospital admissions, obtained from data collected by a medical insurance group, was used by Sterling *et al.*[27] to study the effects of air pollution on hospitalization of about 10,000 individuals in Los Angeles. Certain diseases considered to be relevant to air pollution, such as allergic disorders, inflammatory disease of the eye, acute upper respiratory infections, influenza, and bronchitis, were found to be related to daily concentrations (measured as daily average minimum and maximum values) of oxidants, carbon monoxide, sulfur dioxide, oxides of nitrogen, ozone, oxidant precursor, and particulate matter. Measurements were taken at eight different stations five to ten miles apart from March to October. The analyses showed significantly larger admission rates on those days among the highest third of sulfur dioxide pollution than on those days among the lowest third. Sulfur dioxide concentrations during the period of the study averaged less than 0.015 ppm ($\sim$45 μg/m^3). Concentrations on days of highest pollution were not reported. The method of analysis generally is new and complex, and the author noted the need for additional time periods to be studied to take into account the possibility of seasonal variations. The analysis must also be extended to other cities.

McCarroll *et al.*[24] studied residents of a New York City housing project, using weekly questionnaires. Exact levels of sulfur dioxide which could be used for establishment of air quality criteria were not given; however, the data indicate that sulfur dioxide rather than particulate matter was associated with eye irritation. Symptoms of cough were also shown to be related to air pollution but were not well differentiated between association with particles and sulfur dioxide. The particular time-series analysis used with these data is not well known, and the biases inherent in its use have not been fully determined.

Results also obtained at Rotterdam have shown that when the SO_2 concentration rose for 3 to 4 days from about 300 $\mu g/m^3$ to 500 $\mu g/m^3$ (0.11 ppm to 0.19 ppm),* the number of admissions into hospitals for respiratory tract "irritation" rose, especially in older individuals.[7]

b. Geographical Variations in Mortality

1. *Studies Based on Available Data.*—Mortality and morbidity statistics each have advantages as well as disadvantages. Records of illness should be more fruitful in defining subtle effects, since illness precedes death and since all illness does not result in death. Mortality statistics are collected in every country and are available for quick tabulation. Unfortunately, the quality of mortality statistics varies. One of the problems is that, with the present system of tabulating mortality data, a single cause of death must be selected and coded, even though more than one cause may be involved in the death. The single cause of death designated (e.g., specific chronic respiratory disease) depends largely on the judgment of the attending physician and has little, if any, relation to epidemiologic use. While contributing causes of death appear on the death certificate, they are not reflected in summary tabulations. The coding of only the "underlying" cause of death minimizes the importance of such diseases as emphysema which often appear on the death certificate as contributory or associated causes of death.[28]

Almost all studies of the effects of long-term exposure on death rates compare the rate in one area with that in another. Mortality as well as morbidity studies are hampered by the possibility of differences other than air pollution existing between the areas, such as social class, occupation, age, and sex composition of the population, and cigarette smoking. Assuming that almost all deaths are recorded and tabulated, comparison of total mortality rates (i.e., deaths from all causes) obviates the bias of diagnostic selection, but does not lessen the chances of other associated factors having caused the difference.

Buck and Brown[29] reported in 1964 the relation of standardized mortality ratios for the 5-year period 1955–1959 to four variables: daily smoke and SO_2 concentrations for March 1962 (presumed representative of the study period), population density in 1961, and a social index of 1951. The studies involved populations in 214 areas of the United Kingdom (19 London boroughs, 40 county boroughs, 70 other boroughs, 61 urban districts, and 15 rural districts).

Statistical analysis indicated that bronchitis mortality had a significant positive association with both the smoke and SO_2 concentrations encountered in these residential areas, and also with social index. The standardized mortality rates for lung cancer were not, in general, significantly associated with smoke or SO_2 concentrations in the residential areas. Examination of the tables given by Buck and Brown suggests that the excess of bronchitis mortality occurred for classes of area where the average daily smoke and SO_2 concentrations both exceeded 200 $\mu g/m^3$.* Although smoking habits were reviewed and were apparently uniform from area to area, occupational and domestic indoor environmental exposures were not considered. The pollutant values selected for the correlations did not cover the same time periods as the mortality figures. The associations of bronchitis mortality with smoke and sulfur dioxide were about equal, and the effects of the two pollutants cannot be separated. Variations in smoking habits did not seem to account for much of the mortality variation.

*** SO_2 is converted from ppm to $\mu g/m^3$ in the Dutch reports by using the equivalency, 2,700 $\mu g/m^3 = 1$ ppm. This report uses 2,860 $\mu g/m^3 = 1$ ppm whenever possible.**

*** 200 $\mu g/m^3$ of SO_2 is equivalent to 0.070 ppm.**

In addition to studies of acute variations in mortality in relation to air pollution indices, Burgess and Shaddick[11] studied mortality rates in London and their relationship to smoke, sulfur dioxide, social class, and place of birth (in or out of London). Average levels of sulfur dioxide did not vary much among the areas, ranging from 190 $\mu g/m^3$ to 315 $\mu g/m^3$ (0.07 ppm to 0.11 ppm); sampling was performed at seven widely scattered points every 24 hours during normal winter weather and every 4 hours during fog periods. A significant relationship between lung cancer or bronchitis mortality and smoke or sulfur dioxide was not demonstrated. Births in London and low social class (occupational stratification) were both associated with higher rates. The differences in sulfur dioxide levels are small among the different areas, and it is difficult to believe that, even if sulfur dioxide were a fairly strong causal factor, any significant differences could be shown by this method. What appear to be the same data, slightly rearranged, are reported by Gore and Shaddick[9] with the same basic conclusions.

In a similar study carried out a few years prior to the above, Pemberton and Goldberg[30] compared bronchitis death rates in persons over age 45 in 33 county boroughs of England and Wales for which air pollution data were available. Two indices of social conditions were used: (1) the number of persons per room, and (2) an estimated percentage of households with income exceeding a certain level. Counties from urban and industrial areas were included. Significant correlations were found with bronchitis death rates and sulfur dioxide pollution, but not with "total solids." The association was true for male but not for female bronchitis rates. No significant correlation was found between the two indices of social class and sulfur dioxide pollution. No evidence was given for smoking differences, and no specific sulfur dioxide levels were given that would allow establishment of ambient air quality.

Gorham[31] found a correlation between the crude pneumonia death rate and atmospheric sulfate deposits in the 53 counties of England and Wales. The original data were not given, and the rates were not corrected for age differences or social class; the study can therefore only be suggested until confirmed by more accurate methods.

Other studies[32, 33] have confirmed an association in Great Britain of bronchitis death rates with general indices of air pollution, and an equally strong association in general holds up when various measures of social class are held constant by statistical means. Lung cancer mortality is consistently associated with measures of population density and cigarette smoking, but not with general indices of air pollution.

2. *Special Studies Involving the Collection of New Data.*—In 1964, also, Wicken and Buck[34] reported on a study of bronchitis and lung cancer mortality in six areas of Northeast England, one in Eston, another in Stockton and four in rural districts. The deaths covered the period 1952 to 1962. The survey of decedents with cause of death from bronchitis or lung cancer was matched against the survey of decedents with cause of death from nonrespiratory disease controlled for age and sex; the basis for the diagnostic classifications was not stated in the report. Personal interviews were carried out with next of kin. Personal interviews of a random sample of households were also conducted to obtain sex, age, smoking habits and occupation of the population at risk, the living population. The survey of decedents was carried out between January and October 1963; the survey of the living population was carried out between December 1963 and March 1964. Smoke and sulfur dioxide concentrations were measured in the Eston urban district. One year's aerometric data were obtained. The study was excellent in principle though, unfortunately, sulfur dioxide and particulate values were available only for the Eston urban district.

Eston, itself, as a sub-study, was subdivided into North Eston and South Eston. North Eston contains or lies near heavy industrial plants, whereas South Eston is a residential area. During the period May 1963 to April 1964, mean weekly observations of the sulfur dioxide and smoke concentrations were carried out in two sites in North Eston and one station in South Eston.

The sulfur dioxide value in North Eston on the yearly average was 115 $\mu g/m^3$ (0.040 ppm) and for South Eston it was 74 $\mu g/m^3$ (0.026 ppm). Smoke values were 160 $\mu g/m^3$ and 80 $\mu g/m^3$ for North and South Eston respectively. The deaths studied occurred between 1952 and 1962. Adjustments were made for differences in age composition, smoking habits and social class, and these were insufficient to explain the differences in lung cancer and bronchitis mortality rates between the two localities. Occupational exposure to pollution was then taken into account in the analysis. The conclusion was that there is an association between the degree of air pollution and the incidence of lung cancer and bronchitis mortality in the two areas of the Eston urban district. Though both sulfur dioxide and smoke values and concentrations are furnished in the report and the effects apparently cannot be separated, Brasser *et al.*[7] apparently have used the sulfur dioxide concentration as the more relevant measure of this study.

In a later study by Burn and Pemberton[35] the community of Salford was classified into three pollution areas according to Table 9–2. Five sampling stations in the area were employed. Despite the closeness of the ranges of values, a high rate of bronchitis mortality, of lung cancer mortality, and of deaths from all causes, was observed in the high compared to the lower pollution wards. It appears (see Section C–4) that there was also an increased rate of bronchitis morbidity in the highly polluted wards.

Winkelstein *et al.*[36–38] divided the city of Buffalo into three zones based on a network of 21 air sampling stations operated for a 2-year period. The city was also divided by census tracts into five economic levels based on median family income reported from the U.S. Census. Although the method by which the air pollution zones were determined is not clearly described, in the high zone sulfation levels were greater than approximately 0.45, in the intermediate zone, 0.30 to 0.45, and the low zone less than 0.30 (all units $mg/cm^2/30$ days). Total mortality from all causes for men aged 50 and over showed no association with sulfation within each economic stratum. Deaths from chronic respiratory diseases, including asthma, bronchitis, chronic interstitial pneumonia, bronchiectasis, and emphysema, could be examined only for white males aged 50 to 69 because of insufficient numbers. Only in the second economic level (income $5,175 to $6,004) were there more than five deaths in each air pollution level. Within this income group a clear gradient in mortality for chronic respiratory disease was seen from low to high air pollution, with the biggest difference being that between the moderate and high pollution areas. In the first (i.e., lowest) economic level (income $3,005 to $5,007), mortality for chronic respiratory disease was higher in the "high" oxides of

Table 9–2. POLLUTION LEVELS IN SALFORD. (SEASONAL DAILY AVERAGES)[35]

Pollution area classification	Smoke, $\mu g/m^3$		SO_2, $\mu g/m^3$ (ppm in parentheses)		Deaths $\frac{\text{observed}}{\text{expected}} \times 100$		
	Winter	Summer	Winter	Summer	All Causes	Bronchitis	Lung Cancer
High	680	270	715 (0.25)	255 (0.09)	106	128	124
Intermediate	490	170	460 (0.16)	200 (0.07)	100	97	84
Low	450	170	340 (0.12)	145 (0.05)	90	52	79

Note: The data in the original report show SO_2 concentrations in parts per hundred million (pphm).

sulfur area than in the "intermediate" pollution area. No consistent gradient was found for lung cancer deaths.

As the authors point out, since the only association between sulfation and respiratory disease mortality was in the lowest economic classes (first and second classes), and these classes were likely to have increased occupational exposures to air pollution, the relationship might be due to an intervening occupational variable. However, Reid has called attention to the likelihood of air pollution affecting the lowest socioeconomic group most.[39] This may be ascertained by a study which can measure effects on the lowest socioeconomic group in low, moderate, and high pollution areas. As in most community studies the effect of differences in smoking habits was not known in the Buffalo study. Also, the study did not take account of the ethnic background of the decedents, although Winkelstein's analysis of mortality from gastric cancer as related to particulate levels attempted to do this.

From an aerometric standpoint, one of the most extensive studies of variations in mortality and morbidity within a city was the work of Zeidberg *et al.* in Nashville, Tennessee.[40, 41] Aerometric data were collected at 123 sampling stations in a grid across the city. An area of "high" SO_2 concentrations was defined which had a geometric mean annual 24-hour level of 0.01 ppm ($\sim 30\ \mu g/m^3$) or more. The "low" SO_2 area had 0.005 ppm ($\sim 15\ \mu g/m^3$) or less. The high sulfation area had 0.351 mg $SO_3/100\ cm^2$-day or more as an annual geometric mean. Areas of high, moderate, and low pollution were also defined by soiling index, annual dustfall, and suspended particulate matter. All codable deaths registered between 1949 and 1960 (32,067) were then distributed among census tracts rated according to high, moderate, and low pollution levels, and upper, middle, and lower economic classes, and then further coded by age, sex, race, and underlying cause of death. In the study, account was not taken of smoking habits of the deceased; also, the "middle class" group covered a relatively large segment of the decedents. Standardized mortality ratios (for total respiratory disease, and for pneumonia, influenza, bronchitis, emphysema, tuberculosis, and lung and bronchial cancer) were then related to the pollution indices obtained during 1959. The statistically significant mortality increases were those for all respiratory diseases related to sulfation and soiling; lung and bronchial cancer mortality, and bronchitis and emphysema mortality were not clearly related. "High" pollution in these studies for soiling referred to more than 1.1 coh unit/1,000 linear feet. A later paper [42] derived from the same study period analyzed infant and fetal death rates between 1955 and 1960. For white infant mortality, significant regressions were obtained for sulfation; dustfall (alone, or as an interaction variable) was the most frequently related variable.

In summary, the results of this analysis of long-term exposure studies indicate effects which would coincide with Level III of the World Health Organization's "guides to air quality."[25] Level III is defined as "concentrations and exposure times at and above which there is likely to be impairment of vital physiologic functions or changes that may lead to chronic diseases or shortening of life."

c. *Geographic Variations in Morbidity—Special Studies*

It has been postulated that the study of records of illness rather than mortality should be more fruitful in defining subtle effects, since morbidity is an earlier and more sensitive index of deviation from normal health. A much larger insult must presumably be given to the body to cause death than to cause illness. Routinely collected morbidity data are, however, not generally available. Data may occasionally be obtained from group insurance plans, hospital admission records, or existing school records. Since such data are usually not collected in a uniform, precise manner, most morbidity studies require expensive and time-consuming field surveys with questionnaires or actual medical examinations of the subjects.

Morbidity studies of adults involving long-term exposures are frequently not as useful as desired, due to the presence of complicating factors such as occupation and smoking. Accordingly, Anderson has recommended[43] that children and housewives be used to determine the health effects of air pollution.

A study was conducted by Petrilli *et al.*[44] in Genoa, Italy, which followed Anderson's recommendation. This intensive survey of community respiratory morbidity was actually a set of epidemiologic studies to determine the air pollution effects in Genoa. The subjects studied in one of the research projects were women of 65 years of age, nonsmokers who lived for a long period in the same area and who had no industrial experience. Economic and social conditions in the areas of residence were considered as well as the levels of pollution in these areas of residence. Air pollution was monitored in Genoa for 10 years (1954–1964) at 19 sites covering the suburban area, the residential center, and the industrialized area. Sulfur dioxide was measured by a technique analogous to the volumetric method of the English Department of Scientific and Industrial Research. Carbon monoxide was also monitored. A retrospective survey was carried out using the M.R.C. questionnaire with slight modifications. As has been noted, several different epidemiologic studies appeared to have been conducted. Consideration was taken in the sample selection of age, length of residence in the same area, presence of cardiovascular disease, living habits, previous working habits, etc., in the design of the study. Morbidity indices were calculated for 1961 and 1962 for the population which received free medical care from the municipality and therefore was under continuous medical observation. Prevalence and incidence rates were calculated for symptoms such as cough, sputum, dyspnea, rhinitis, and recent and past respiratory diseases. The frequency of these respiratory symptoms was much greater in the central residential area than in the suburban residential area, even though the annual means of sulfur dioxide in the two areas were relatively close; the pattern for rhinitis was somewhat erratic.

Sulfur dioxide measurement in the cleanest area, that is, the suburban residential district, was 80 $\mu g/m^3$ (0.028 ppm)* with the winter value being 85 $\mu g/m^3$ (0.030 ppm) and the summer value 55 $\mu g/m^3$ (0.019 ppm). The residential center area had an annual average value of 105 $\mu g/m^3$ (0.037 ppm), with a winter average of 115 $\mu g/m^3$ (0.040 ppm) and a summer average of 85 $\mu g/m^3$ (0.030 ppm). The industrial area averaged 265 $\mu g/m^3$ (0.093 ppm) for its annual mean with a winter average of 315 $\mu g/m^3$ (0.11 ppm) and a summer average of 230 $\mu g/m^3$ (0.080 ppm).

The study showed a striking correlation between frequency of symptoms and the diseases of the respiratory tract and air pollution levels. In the suburban residential area, the indices were almost always significantly lower for the data collected and particularly for the frequency of cough, sputum, dyspnea, rhinitis, and recent respiratory disease, especially bronchitis. There was usually a gradient between the suburban area and the industrial area. What was especially significant was the finding of differences generally in the prevalence of respiratory diseases during the summer in the industrial area as compared with the moderate and low pollution areas. This should contribute to our knowledge regarding the relationship between different climatic conditions and the health effects of air pollution. It has been postulated that it is usually in the winter that air pollution of the reducing type exerts the most deleterious effects.

The study also examined a number of areas in the city. Data for the seven districts in the town of Genoa showed a very significant correlation (r = 0.98) between the frequency of bronchitis and the annual mean of sulfur dioxide levels. A nonsignificant correlation between the frequency of bronchitis and suspended matter and dustfall was observed in the study. There was a suggested relationship between winter temperature and bronchitis, but this was further reduced when adjustment was made for air pollution.

What is particularly significant about this study is that the differences in respiratory disease were found between areas with an annual mean of 105 $\mu g/m^3$ (0.037 ppm) and one of 80 $\mu g/m^3$ (about 0.028 ppm). Also highly significant was the observation that effects of air pollution were found in the summer prevalence of respiratory disease. Very sharp differences were found within a community which is characterized by maximum sulfur dioxide levels below an annual average of 300 $\mu g/m^3$ (0.10 ppm). Finally, the very striking differences between the area with low levels of pollution and the industrialized areas were noted and should lend support to the results of studies in other areas.

---

* SO_2 values in this study were originally reported in ppm.

Toyama,[45] in a comprehensive study of air pollution and its health effects in Japan, charted the age-standardized morbidity rates (per thousand) secured by interview survey in 1961, and described a gradient of respiratory disease morbidity from the highly industrialized (and presumably polluted) areas to the rural areas of Japan. Further, the pulmonary disease morbidity ratio was higher in the industrialized, polluted areas than were the ratios for other disease groupings. This gradient was not noted for cardiovascular diseases nor for gastrointestinal diseases. Unfortunately, specific pollutant concentrations were not clearly indicated to accompany these data on morbidity. The age-standardized morbidity rates per thousand for several cities in Japan are shown in Figure 9–3.[45]

Pulmonary function and respiratory symptoms were compared in population areas of high (95 $\mu g/m^3$) and low (25 $\mu g/m^3$) SO_2 pollution (annual means of 0.034 ppm and 0.009 ppm respectively)* (measured by H_2O_2 absorption, H_2SO_4 titration) in Port Kembla, Australia.[46] Coh values in the more polluted and in the cleaner area, averaged over one year, were 2.7 and 1.3 respectively. Since the pollution in this area was due to a single large source, pollutant levels fluctuated widely. One peak reading of over 13 ppm was observed, and daily averages ranged up to 17 times the annual mean. Chronic bronchitis rates for men over 55 were higher in the polluted area, but rate differences for other age groups and for women were not statistically significant between the areas. A higher incidence in the polluted area was also found for a variety of respiratory symptoms such as head colds, wheezing, and chronic cough. The effects were said to have disappeared when pollution was reduced by using a higher stack at a smelter.[47] Adjustments were not made for differences, which were known to exist, in age and smoking between the two areas. In addition, the heavily polluted area was of lower socioeconomic class: only 3.2 percent of its population was in professional and administrative occupations, and 10.2 percent were laborers as compared to 10.2 percent professionals and 2.4 percent laborers in the clean area. A difference of over one inch in average

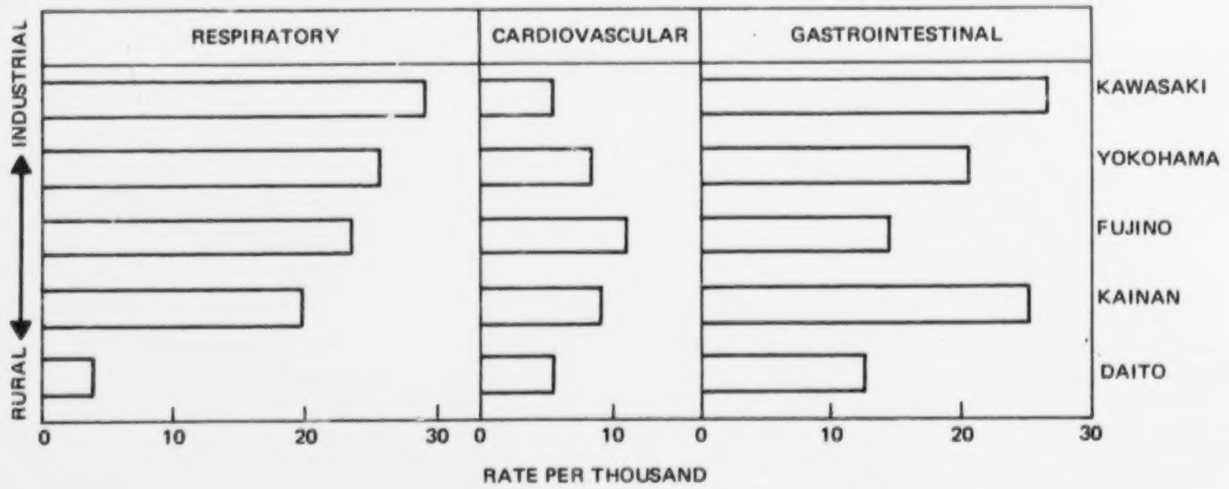

Figure 9-3. Age-Standardized Morbidity Rate per 1,000 for Three Diseases in Japan.[43]

The mortality rate for respiratory diseases shows a clear correlation with pollution levels. There is no such clear correlation for cardiovascular or gastrointestinal disorders.

---

* SO_2 values in this study were originally reported in ppm.

height of the population in the two areas also indicates the two populations were not completely comparable. No significant differences that could be related to air pollution were found in respiratory function measured as $FEV_{1.0}$. In Port Kembla, SO_2 concentrations are highest during the summer, rather than winter, so that this study is not biased by the question of whether the effects are due to pollution or low temperature, as has been the case in many of the British and American epidemiologic studies. Notwithstanding the stated deficiencies of the study, the finding that respiratory symptoms disappeared when pollution was reduced would appear to confirm an effect of sulfur dioxide at the level found originally in the old high pollution area of Port Kembla, if no other change took place which could account for the reduction.

In 1965, Holland *et al.*[48, 49] reported on their study of the prevalence of chronic respiratory disease symptoms and performance of pulmonary function tests of outdoor telephone workmen in London, in rural England, and on the east and west coasts of the United States. Types of occupational exposure, use of cigarettes, and socioeconomic matching were considered. The annual mean concentration of suspended particulate matter in the British exposures, both rural and in London, was approximately 200 $\mu g/m^3$. For sulfur dioxide, the mean concentrations were: 285 $\mu g/m^3$ (0.1 ppm)* in London and in rural areas of England, 55 $\mu g/m^3$ (0.02 ppm). Persistent cough and phlegm and chest illness episodes were 1.4 times as frequent in men aged 40 to 49, and 2.2 times as frequent in men aged 50 to 59, in the London area as in rural England. Increased sputum volume was more frequent and pulmonary function was poorer in London than in the rural areas. These differences held, even when the data were standardized for smoking.

In an earlier study, conducted in 1960 and 1961 following a number of related studies yielding consistent results, Holland and Reid [50] reviewed respiratory symptoms, sputum production, and lung function levels in mail van drivers and vehicle maintenance men in central London and mail van drivers and engineering workers also driving vans in and around three county towns in Southern England. The conduct of the study was exemplary. A standardized questionnaire was used as were trained interviewers. Because of the design of the study, socioeconomic factors were the same, the occupational exposures were homogeneous, and corrections were applied for smoking. There were some physique differences in the rural areas, and allowances were made for these in the statistical evaluation. Unfortunately, no comprehensive indices of air pollution were available for the three towns. However, mean sulfur dioxide readings expressed as sulfate per 100 cm^2 lead oxide were available for two stations in St. Pancras near the London survey center and two stations in Gloucester, one of the three towns. Brasser *et al.*[7] have furnished the following SO_2 values: London (St. Pancras) summer average 100 $\mu g/m^3$ (0.035 ppm), winter average 500 $\mu g/m^3$ (0.17 ppm) and Gloucester, Peterborough and Norwich 75 $\mu g/m^3$ (0.026 ppm) and 200 $\mu g/m^3$ (0.070 ppm) respectively for summer and winter averages.

Between the age of 50 and 59, the London men had more frequent and more severe respiratory symptoms, produced more sputum, and had significantly poorer performance on lung function tests. Dyspnea of grade 3* or more was more than four times as frequent in London as in the county towns among men 50 to 59 years of age; this is a highly significant difference. The authors concluded that the relatively high level of pollution in London was the major reason for the excessive frequency or severity of chronic bronchitis in London.

In a group of Canadian veterans studies by Bates *et al.*,[51] a relationship between air pollution and both bronchitis and pulmonary function measurements has been reported. There are, unfortunately, inadequate data on the levels of smoke, sulfur dioxide, and other pollutants, in the four Canadian cities compared, to derive specific relationships between the levels of these pollutants and the prevalence or exacerbation of disease or the deterioration of pulmonary function. However, there is an association in the "dirty" cities (Montreal and Toronto) versus the "clean" cities (Halifax and Winnipeg) of increased prevalence and severity of bronchitis, and poorer pulmonary function performance.

---

* SO_2 values in this study were originally reported in ppm.

* The author grades dyspnea [49] according to five levels; grade 3 represents dyspnea when walking even at a slow pace on level ground, but not to the extent that it is necessary to stop for breath.

One of the most detailed and carefully designed American studies was carried out by Anderson and Ferris in Berlin, New Hampshire, and Chilliwack, British Columbia.[52–54] In each town a carefully drawn, random sample of the population was given standard pulmonary function tests and a standard questionnaire for respiratory symptoms. In Berlin, the sulfation rate for August and September 1960 averaged 426.0 $\mu g/100\ cm^2$-day while in Chilliwack the sulfation concentration for August and September 1963 averaged 50.3 $\mu g/100\ cm^2$-day. This was converted to an equivalent sulfur dioxide value for Berlin of 35 $\mu g/m^3$ (0.012 ppm)* based on readings for one station. No information is provided to show how the Chilliwack sulfation values were converted to an SO_2 equivalent of 2.85 $\mu g/m^3$ (0.001).* Soiling values were virtually identical; coh values averaged 0.5 for Berlin and greater than 0.5 for Chilliwack. The prevalence of chronic respiratory disease was initially greater in Berlin, but adjustments for differences in age and in cigarette smoking eliminated all the differences in prevalence. However, even after correction for age, height, sex, and smoking habits, the Berlin residents had slightly lower results on pulmonary function tests ($FEV_{1.0}$ and Peak Expiratory Flow Rate). This difference might be due to the different climates, or to a slightly lower socioeconomic class in Berlin, or to ethnic differences between the two towns, or to occupational exposures. There were also studies comparing areas within Berlin, and no difference in respiratory symptoms or pulmonary function could be found between an area where the sulfation rate averaged 610 $\mu g/100\ cm^2$-day (2-month period) and another area of the town averaging 255 $\mu g/100\ cm^2$-day for the same period.[52, 53, 55]. As in the two-town study, the disease rates in the two areas of the town were strongly affected by differences in the proportions of the populations who were cigarette smokers. The authors correctly note the limited possibility of ascertaining effects, due to the very low pollution values and the movement of people with respiratory disease to the even less polluted areas of Berlin.

A study[56] comparing two areas exposed to pollution from a single source was undertaken in the Seward-New Florence area of Pennsylvania. The sulfation rate was 6.2 times greater in Seward (3.7 mg $SO_3/100\ cm^2$-day) than in neighboring New Florence, while suspended particulate matter concentrations were 1.4 times greater. These were 151 $\mu g/m^3$ and 109 $\mu g/m^3$ respectively. During the study period from October 1959 to April 1960, the average 24-hour SO_2 measurement was 255 $\mu g/m^3$ (0.09 ppm)* in Seward, compared to less than 30 $\mu g/m^3$ (0.01 ppm) in New Florence, both by the West-Gaeke method. Slightly higher values for airway resistance were found in Seward even after correcting for differences in height and age. No adjustment was made for other factors which may have affected airway resistance, such as smoking and occupation. The difference in body height is also difficult to interpret.

The Nashville air pollution study reviewed total morbidity in relation to air pollution.[40] The morbidity data were obtained by trained interviewers in a questionnaire survey of sample households in the city. The aerometric data used for the survey were the same as those used for the morbidity study. An area of "high" SO_2 concentration was defined as one which had a geometric mean annual 24-hour level of 30 $\mu g/m^3$ (0.01 ppm)* or more. The "low" SO_2 area had 15 $\mu g/m^3$ (0.005 ppm) or less. The high sulfation area had 0.351 mg $SO_3/100\ cm^2$-day or more as an annual geometric mean. Areas of high, mod-

---

** SO_2 values in this study were originally reported in parts per billion (ppb).

* SO_2 values in this study were originally reported in ppm.

erate, and low pollution were also defifined by soiling index, annual dustfall, and suspended particulate matter. As expected, when the data were analyzed by socioeconomic class, higher illness rates were reported from the lower class households, and there was a strong tendency for persons of lower social class to be resident in areas of high air pollution.

Since only the large middle class were present in all pollution zones, analyses controlled for socioeconomic status were limited to it. A direct correlation between illness rates from all causes and pollution could not be shown with any consistency except for persons 55 years and older (both sexes). A decline in morbidity rates from high to low pollution areas could be shown for this group with the soiling index and SO_2 concentrations as pollutant variables, but no consistent decline with sulfation, dustfall, or suspended particle concentrations was seen. There was no correlation between pollution indices and morbidity rates for cancer, respiratory, or gastrointestinal diseases. Attempts were made to minimize the effect of occupational exposure by studying females keeping house. In this group, a small but consistent gradient in illness from all causes correlated with air pollution indices. No consistent findings could be found for working females from all social classes combined. Because of the possible wide variations within the large group called "middle class," the study may not have taken socioeconomic status wholly into account. The lack of information on cigarette smoking is another deficiency. The morbidity differences that are shown are most striking between the "high" pollution group versus the "moderate" and "low."

d. Morbidity—Incapacity for Work

Dohan[57, 58] compared respiratory morbidity in hourly employees in five United States cities by use of insurance and personnel statistics of a large electronics company. Hourly employees received insurance payments after the seventh day of illness provided that a physician's certificate was received. For the 3 study years, the respiratory absentee rates showed a direct relationship to suspended particulate sulfates. The latter concentrations ranged from a low of 7 $\mu g/m^3$ in Cincinnati to about 14 $\mu g/m^3$ in Camden and Woodbridge, New Jersey, and in Indianapolis, to a high of 20 $\mu g/m^3$ in Harrison, New Jersey. A breakdown by type of respiratory disease showed miscellaneous respiratory infections, influenza, and bronchitis to be most directly related to the sulfate levels (see Figure 9–4). Pneumonia, asthma, and sinus disease were not significantly correlated. The incidences of gynecologic problems, urinary tract disease, and appendicitis were not related to several indices of air pollution, including sulfate; nonoccupational accidents did, however, increase progressively with sulfate, as did the respiratory diseases. In addition, neuropsychiatric diseases were highest in the three cities with highest pollution indices. The correlation between all indices of air pollution and accidents and mental illness may indicate that secondary ef-

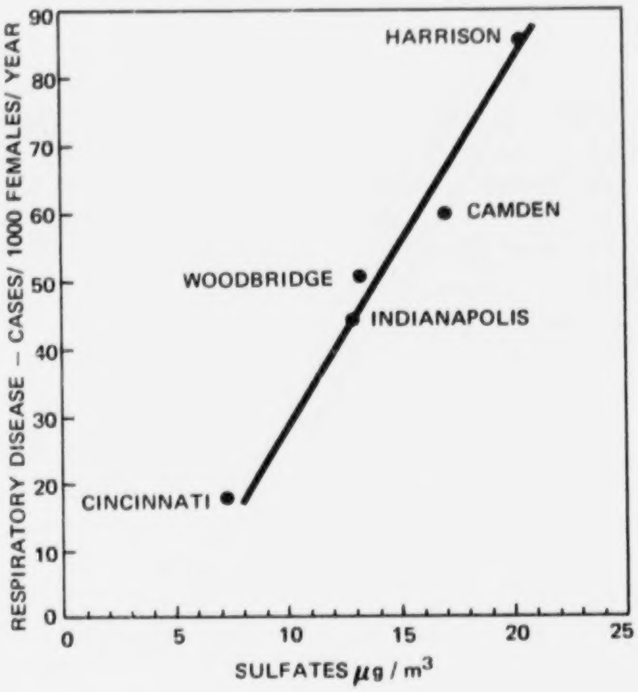

Figure 9-4. Incidence of Respiratory Disease Lasting More than Seven Days in Women Versus Concentration of Sulfates in the City Air at Test Sites.[54]

The incidence of respiratory disease lasting more than seven days in this group is directly related to concentrations of suspended sulfates in the cities shown.

fects of air pollution, such as reduced visibility and gloomy weather, have a measurable effect on health and welfare, or that the results are too *nonspecific* to attach causal significance.

Age differences were rather marked among the cities, yet age standardized rates were not calculated. Without detailed calculations it is not possible to state whether differences such as only 0.2 percent of the Cincinnati women being over age 45 compared to 35 percent of Camden, New Jersey, women would significantly affect the rates. Differences in social class and standard of living were probably not great, since all subjects were hourly employees of the same company. Differences in patterns of illness reporting and acceptability of different diagnoses within each city are difficult to assess, but presumably were minimized by limiting the study to absences of 8 days and more. Other possible differences, such as weather and climate in the cities, exposure to toxic materials, plant morale, and air conditioning were discussed by the authors and judged by them not to be of major importance. No data are available on differences in cigarette smoking, or plant rules regarding smoking at work, in the different locations. A unique and strong factor of this study is that other pollutants such as total suspended particlate matter, benzene-soluble organics, and certain trace metal concentrations did not correlate with the respiratory disease rates. The findings of this study need to be confirmed.

During 1961–1962 a study of the incidence of incapacity for work was conducted by the British Ministry of Pensions and National Insurance.[59] The population covered was representative of the working population of England and Wales. Rates of sickness absence for bronchitis, influenza, arthritis, and rheumatism were related to indices of pollution. There was a significant correlation between bronchitis incapacity in middle-aged men (35 to 54) and the average seasonal (October through March) levels of smoke and sulfur dioxide in high-density residential districts, based on 24-hour measurements. For Greater London, there was a significant correlation between bronchitis incapacity and *both* smoke and sulfur dioxide for all age groups taken together, and for men aged 35 to 54 and 55 to 59. It is interesting that there was also more incapacity from arthritis and rheumatism in areas having heavy smoke pollution. Influenza incapacity was greater in those areas with higher pollution levels over Great Britain as a whole but not within the Greater London conurbation, nor was there in this latter area any association between pollution and psychosis or psychoneurosis. The lowest bronchitis inception rates related to smoke levels between 100 $\mu g/m^3$ and 200 $\mu g/m^3$ and to sulfur dioxide concentrations between 150 $\mu g/m^3$ and 250 $\mu g/m^3$ (0.053 ppm and 0.081 ppm). The highest values related to concentrations of 400 $\mu g/m^3$ smoke and 400 $\mu g/m^3$ (0.14 ppm) sulfur dioxide.

Burn and Pemberton [35] compared areas of high and low pollution in Salford, England. Pollution data were obtained from five standard D.S.I.R. instruments operating for 2 years, 1958 and 1959. The aerometric data were obtained for 24-hour periods. Average daily sulfur dioxide concentrations during the winter months in the more polluted areas were approximately 715 $\mu g/m^3$ (0.25 ppm)* compared to 285 $\mu g/m^3$ (0.10 ppm) in the cleaner areas (measurement method not clear). Smoke pollution was also considerably higher in the polluted areas. Bronchitis morbidity, as measured by incapacity certificates from the Ministry of National Insurance, was higher for the study group (men aged 45 to 64) in the polluted areas.

Brasser *et al.*[7] have reanalyzed the Salford data without indicating why this reanalysis was necessary. They prepared new isopleths and concluded that the correlations between absenteeism for bronchitis and pollution levels are considerably higher than originally was presumed by the investigators themselves. The population 45 to 64 used for denominators of the rates had to be estimated by assuming that each district had the same proportion in that age group as the town as a whole. In addition, large differences in socioeconomic class probably existed between the areas, but no adjustments were possible. Differences in occupation may also have af-

---

*** SO_2 values in this study were originally reported in pphm.**

fected incapacity rates. No data on cigarette smoking were available.

Results obtained in The Netherlands have also shown that when the SO_2 concentration rose for 3 to 4 days from about 300 $\mu g/m^3$ to 500 $\mu g/m^3$ [0.11 ppm to 0.19 ppm]*, absenteeism at both Rotterdam and Vlaardingen increased by at least 30 percent; in certain groups (e.g., those over 45 years of age) the increase was as large as 50 percent to 100 percent.[7]

Verma *et al.*[60] presented information on illness absences in relation to air pollution. Illness data for the employees (males and females, ages 16 through 64) of a metropolitan New York insurance company were obtained through the records of the personnel department. They included medical history, X-ray information, and laboratory results obtained by the medical department of the company, and were classified by absences due to respiratory illness and to nonrespiratory illness. Mean daily concentrations of air pollutants and meteorologic data were secured from the monitoring system of metropolitan New York: smoke shade, sulfur dioxide, and carbon monoxide content were reported. The data for the 2 years, 1965 and 1966, were examined statistically and several conclusions were reached. There was a strong time dependence and a yearly cyclical behavior; when these factors were removed there remained *no* strong positive relationship between respiratory absence and the pollution variables studied. Respiratory illness absence rates were at their highest level when sulfur dioxide and smoke shade levels were both high on cool days; and a lag effect for respiratory absences was *not* noted.

3. Studies of Children

Comparisons of the prevalence of respiratory disease in areas of varying pollution levels have been made to delineate the roll of air pollution and specific pollutants. A problem common to all the studies is the difficulty in guaranteeing that the areas are similar (except for air pollution) in all factors that might affect the prevalence of disease.

Because studies on adults tend to be complicated by smoking habits, changes of occupation, and changes in address over a period of years, several studies have been directed at effects of air pollution on school children. The advantages of using children for research on the primary etiologic effects of air pollution was first noted several years ago;[61] Anderson has most recently reaffirmed this view.[43] A major element of concern is that deleterious effects on the respiratory system of very young children may have an effect on the subsequent evolution of the chronic bronchitis syndrome in the adult population.

The relationships of respiratory infections in children to long-term residence in specific localities have been studied in England by Douglas and Waller.[62] Levels of air pollution, in terms of domestic coal consumption records, were used to classify four groups; the authors include an evaluation of the validity of this method at the end of their report. The histories of 3,866 children born during the first week of March 1946 were followed until 1961, when the children were 15 years of age. Social class composition of these children did not differ significantly from area to area. Measured concentrations for smoke and for SO_2 in 1962 and 1963 were compared with the earlier prediction of pollution intensity based on the coal consumption data, and indicated an overlap for greater London area of low and moderate groupings; for other areas, the predicted gradient of concentrations was affirmed. Sulfur dioxide varied from about 90 $\mu g/m^3$ (0.031 ppm) in the "very low" group to about 250 $\mu g/m^3$ (0.087 ppm) in the "high" pollution group. Because of the age of the subjects, smoking was apparently not considered in this evaluation. In 1965, 19 percent of the boys and 5 percent of the girls, aged 11 to 13, smoked at least one cigarette a week regularly.[63]

In the Douglas and Waller study, the generation of pollutants in the indoor home environment (e.g., by heating and cooking) was not considered. Interviews were conducted with the mothers when the children were 2 and 4 years of age; information was obtained about upper and lower respiratory illness and recorded hospital admissions for these and other causes. Data about colds, coughs, and hospital admissions were also gathered by school doctors at medical examinations when the children were 6, 7, 11 and 15 years of age. Between the ages of 6-½ and 10-½, special records for causes of school absence exceeding one week were reviewed with the mothers. Families numbering 3,131 remained in the same pollution area throughout the first 11 years of this study. The conclusions of the study were that upper respiratory tract infections were *not* related to the amount of air pollution, but that lower respiratory tract infections were. Frequency and severity of lower respiratory tract infections increased with the amount of air pollution exposure, affecting both boys and girls, and with no differences detectable between children of middle class and working class families. This association was found at each of the examination ages, including age 15. At age 15, persistence of rales and rhonchi (chest noise), possibly the prodrome of adult chronic respiratory disease, was some 10-fold less in the very low pollution area, and a factor of 2 less in the low pollution area than that in the high pollution area. If the 1962-1963 measured concentrations for smoke and SO_2 can truly be extrapolated to the 15-year respiratory illness survey, then these British school children experienced increased frequency and severity of lower respiratory diseases in association with annual mean smoke concentrations ranging above 130 $\mu g/m^3$ and SO_2 above 130 $\mu g/m^3$ (0.046 ppm).

---

* The numbers in brackets are the editor's; an equivalency of 2,700 $\mu g/m^3 = 1$ ppm SO_2 was used to assure consistency with any conversions made by the authors.

The lower respiratory tract findings of Douglas and Waller were confirmed in a study by Lunn *et al.*[64] The patterns of respiratory illness in school children of the age group 5 to 6 were studied by them with reference to residence in four areas of Sheffield. The data were collected during the summers of 1963, 1964, and 1965 in order to minimize effects of winter respiratory illness upon the pulmonary function tests. Mean daily smoke levels measured in each of four areas ranged from 97 $\mu g/m^3$ in the "low" area to 301 $\mu g/m^3$ in the "high" area. SO_2 concentrations were respectively 123 $\mu g/m^3$ (0.043 ppm), 181 $\mu g/m^3$ (0.063 ppm), 219 $\mu g/m^3$ (0.077 ppm), and 275 $\mu g/m^3$ (0.096 ppm) in the four areas, during 1963–1964. Somewhat lower smoke levels were noted the following year, although the gradient between the districts was preserved. However, the sulfur dioxide values were obtained at five schools between October 1965 and May 1966 inclusive. The values were 109 $\mu g/m^3$ (0.038 ppm) and 134 $\mu g/m^3$ (0.047 ppm) corresponding to the "low" pollution area, and then 194 $\mu g/m^3$ (0.068 ppm), 241 $\mu g/m^3$ (0.085 ppm), and 304 $\mu g/m^3$ (0.11 ppm) respectively. Questionnaire of the parents, physical examination, observation for the presence of nasal discharge, examination of the eardrums, and recording of both the forced expiratory volume in 0.75 seconds ($FEV_{0.75}$) and the forced vital capacity (FVC), were completed during each of the summer terms of 1963, 1964, and 1965. Several socioeconomic factors were compared for the various districts; smoking was appropriately disregarded for this age group; internal home environments, or differences in home heating systems, were not reported. The authors conclude that there *is* an association between the levels of atmospheric pollution and chronic *upper* respiratory infections (as indicated by mucopurulent nasal discharge, history of three or more colds yearly, or scarred or perforated eardrum). Further, lower respiratory tract illness (measured by history of frequent chest colds or episodes of bronchitis or pneumonia) was similarly associated. Functional changes, in the form of reduced $FEV_{0.75}$ ratios emerged where there was a past history of pneumonia or bronchitis, of persistent or frequent coughs, or of colds going to the chest. There appeared, therefore, to be a persistence of respiratory dysfunction, even in the absence of high-pollution extant at the time of the function testing. This study clearly demonstrated the association between respiratory illness in infant school children and atmospheric pollution. The lowest "effect" level for smoke and SO_2 was not clearly indicated by this study, but the increased association of "respiratory infections" in school children was detected in areas whose mean daily figures exceeded about 100 $\mu g/m^3$ for smoke and 120 $\mu g/m^3$ (0.042 ppm) for SO_2.

A study for Manzhenko[65] of upper respiratory tract conditions in school children in Irkutsk is difficult to relate to the Sheffield study. However, the higher incidence of respiratory tract conditions and the undefined abnormal X-ray findings in these children's lungs are disturbing evidence of the possibility of an association between serious respiratory disease and residence in a polluted community.

Toyama[45] studied two groups of school children, 10 to 11 years old, in Kawasaki, Japan. Sulfation rates at the school in the more polluted area varied from 0.5 to 1.9 and averaged 0.9 mg/100 cm²-day PbO_2; no sulfation rates were given for area of lower pollution. The children from the more polluted area had a higher frequency of nonproductive cough, irritation of the upper respiratory tract, and increased mucus secretion. The dustfall rate was considerably lower in the less polluted area except for an occasional month when the dustfall in the two areas was almost equal. Whether the effect was due to oxides of sulfur or particulate matter cannot be determined from this study.

4. Studies of Pulmonary Function

Spicer *et al.*[66] studied a group of patients with chronic obstructive airway disease who resided in a small area of downtown Baltimore. Air samplers were placed at the residential area and at the University, where extensive pulmonary function tests were performed. The patients became better or worse in their pulmonary function as a group, suggesting that they were influenced by a factor in their common environment. It was not, however, possible to determine a simple cause and effect relationship with any one pollutant. Subtle changes in the pollutant concentrations and weather conditions may have been responsible.

Shephard *et al.*[67] studied a group of 10 cardiac cripples who were confined to their homes. Air pollution measurements were carried out by sampling equipment placed inside the homes. Pulmonary function tests were repeated at the same hour of the day, 3 times weekly, for 2 months. Although a response to suspended particulate pollution was demonstrated, no consistent changes in pulmonary function were noted with sulfur dioxide levels, measured as total gaseous acid (*sic*). During one inversion period, a 6-hour average of sulfur dioxide concentrations (hydrogen peroxide titremetric method) reached 460 $\mu g/m^3$ (0.16 ppm)*. The indoor readings were considerably lower, the highest indoor concentration being sustained for 6 hours.

Holland *et al.*[49] report decreased performance of the $FEV_{1.0}$ test in London and British rural outside telephone workers compared with their American counterparts. For both groups the FEV was further decreased in relation to smoking intensity. FEV differences within the United Kingdom workers (i.e., London versus rural), may be related to an appreciable extent to the sulfur dioxide concentrations accompanying the particulate levels. A more detailed discussion of the study appears in Section C.

Toyama[45] reported measurements of peak flow rate and total vital capacity performance in Japanese school children in areas of differing air pollution measured monthly; fluctuations were observed in the mean peak flow rates of children attending schools and living in polluted industrial areas, and the variations were smaller for children in cleaner areas. Total vital capacity was not significantly different among pupils of the various schools. There was a substantial difference in peak flow rates between the two school districts at times of highest pollution. When pollution values were lowest, the differences were less. Monthly dustfall was 15 to 70 tons/km²-mo° in the more polluted areas; lead peroxide candle sulfation rates from month to month ranged from 0.5 to 2 mg SO_3/100 cm²-day in the higher polluted area.

In Osaka, Watanabe[22] studied the peak flow rate and vital capacity performances of children in schools enduring differing air pollutant concentrations. It was noted that individual peak flow rates were more markedly decreased in the winter months (September to December 1963) for the school in the highly polluted area than for the school in the low pollution area. Daily mean concentrations of both dustfall and sulfur dioxide concentrations were twice as great in the polluted area as in the unpolluted area.

---

* SO_2 values in this study were originally reported in ppm.

° In the United States, dustfall is measured in tons/mi²-mo.

In the paper by Lunn *et al.*[64] Sheffield school children were shown to have reduced $FEV_{0.75}$ and FVC ratios in the area of highest pollution. The measurements were made during the summer, when pollution levels were low and apparent incidence of acute respiratory infection was diminished, suggesting, in contrast to the Japanese studies referred to above, that there may be persistence of the respiratory function deterioration in relation to residence in the area of high pollution. Mean daily averages measured at a single station were: smoke, 300 $\mu g/m^3$; SO_2, 275 $\mu g/m^3$ (0.096 ppm). In the Port Kembla study [46] mentioned in Section C–2c of this chapter, no significant differences in pulmonary function were noted which could be attributed to air pollution.

The studies relating morbidity and deterioration in pulmonary function to particulate levels cover effects which are also included in Level III of the World Health Organization's "guides to air quality."[25]

5. Studies of Panels of Bronchitic Patients

Lawther [68] related several episodes of acute urban pollution to a worsening of condition in a group of bronchitic patients, well studied in a registry at St. Bartholomew's hospital in London. Changes in their symptomatology were recorded in a daily diary, and acute worsening in significant numbers of the group was associated with daily rises in air pollution above 300 $\mu g/m^3$ of smoke and 600 $\mu g/m^3$ (0.21 ppm)* of sulfur dioxide. Figure 9–5 shows graphically the effects observed on 29 bronchitic patients of high pollution levels in January 1954.

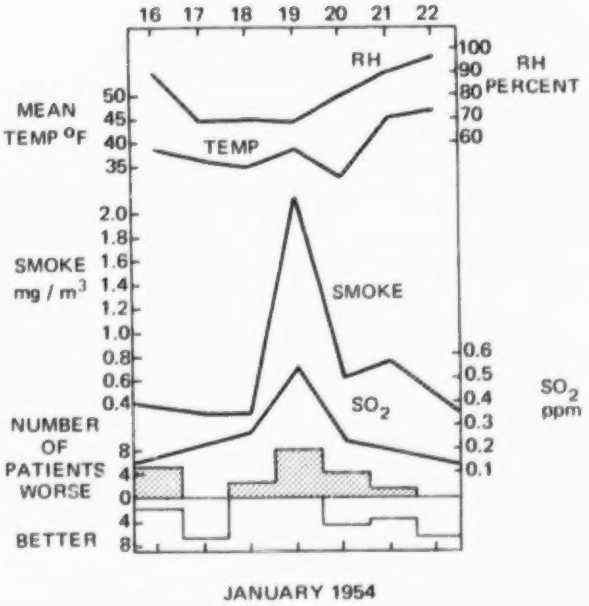

Figure 9-5. Effect on Bronchitic Patients of High Pollution Levels (January 1954).[65,68]

The figure represents the effect on bronchitic patients of increased pollution levels; patients stated whether they regarded their condition as "worse" or "better".

Angel *et al.*[70] reviewed the occurrence of new respiratory symptoms in men working in factories and in offices, most of whom had prior evidence of chronic bronchitis. The study group of 85 men, observed through the winter of 1962–1963, was selected from a group of 1,000 men, age 30 through 59, without apparent classification of either smoking patterns or possible occupational or residential exposure differences. Increased sputum production, deterioration of pulmonary function performance ($FEV_{1.0}$), and the more frequent occurrence of respiratory symptoms classified as "upper" (coryza, influenza, and acute respiratory disease), and "lower" (chest colds, bronchitis, wheezy attacks, pneumonia), were all associated with increases in both smoke and sulfur dioxide concentrations. There was frequently difficulty in defining an exacerbation of disease in those individuals already experiencing chronic bronchitis. During this period, illness peaks (attack rate) may have occurred with weekly mean concentrations of smoke exceeding 400 $\mu g/m^3$ and of sulfur dioxide exceeding 460 $\mu g/m^3$ (0.16 ppm)*; weekly mean concentrations were calculated using the highest daily mean occurring each week at each of 13 locations in the area.

Carnow *et al.*[71] studied over 500 patients with respiratory disease in Chicago during

---

* SO_2 values in this study were originally reported in ppm.

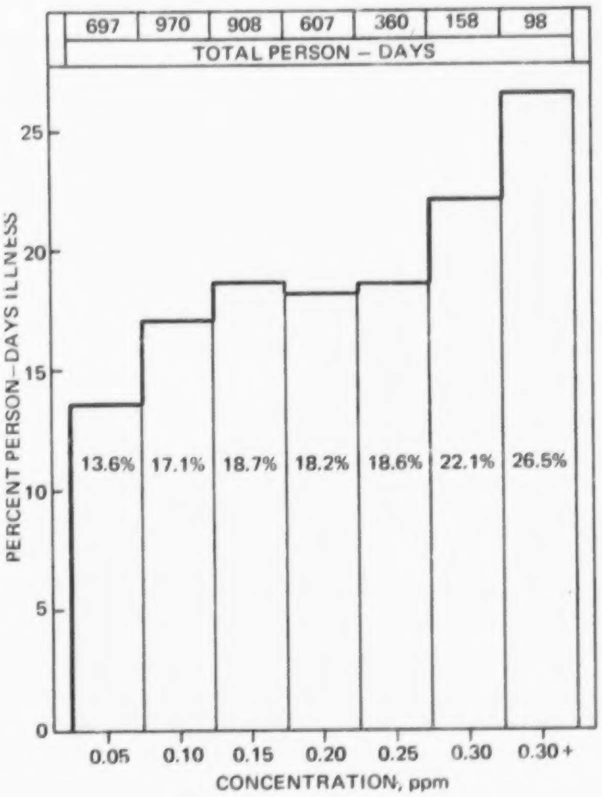

Figure 9-6. Comparison of Person-Days of Acute Illness with Seven Levels of Sulfur Dioxide Exposure in Chicago, in Patients with Severe Chronic Bronchitis (Age 55 or More), for October-November, 1967.

There is an increase in illness for bronchitics over age 55 with increase in sulfur dioxide concentrations. The relationship is with 24-hour SO_2 levels on the day prior to illness.

the winter of 1966–1967. Semidaily averages of sulfur dioxide (West-Gaeke Method) were computed from eight continuous monitoring stations in Chicago for the hours of 6 a.m. to 6 p.m., and for 6 p.m. to 6 a.m. Pollution by particulate matter was also present but not analyzed in this report; the geometric mean for particulates in Chicago during 1964–1965 was 148 $\mu g/m^3$, according to the National Air Surveillance Network (NASN) data.[72] The semidaily averages were used to estimate the 24-hour average concentrations of sulfur dioxide to which the patient was exposed at work and at home. The chronic bronchitics were divided into two groups according to the severity of their disease, and the analyses were kept separate for the two groups. For patients age 55 and over with severe bronchitis, a significant association was demonstrable between the level of sulfur dioxide and person-days of illness. The differences in illness rates in this group for different levels of exposure was most marked for disease on the day following exposure. A marked rise in illness rate was noted for patients exposed to 715 $\mu g/m^3$ (0.25 ppm)* of sulfur dioxide or more. (See Figure 9-6.)

For patients under 55, no differences in illness were noted with exposure, except for the group exposed to 860 $\mu g/m^3$ (0.30 ppm) or more of sulfur dioxide.

For the patients with *mild* bronchitis, illness was not related to air pollution in those age 55 or more, while those under age 55 seem to be worse when exposed to more than 860 $\mu g/m^3$ (0.30 ppm) SO_2. These analyses indicate that groups of persons exposed to higher levels of sulfur dioxide tend to experience higher rates of acute respiratory diseases. However, the group with higher respiratory illness rates could merely happen to live in the more polluted areas. The situation is similar to the findings of most epidemiologic studies which indicate that persons of low social class tend to have high illness rates and also tend to live in more polluted areas. In an attempt to eliminate this class-site factor, further analyses were therefore carried out for the same groups of patients, but this time for single months, using each patient as a control against himself. For each patient, the difference between his mean exposure to SO_2 on days of illness and on days of no illness was computed. The only significant differences were for those age 55 and over with severe bronchitis; no statistically significant differences were demonstrable for the other groups. This tends to show that the association for mild bronchitic in the younger age group mentioned above was more likely due to another factor.

An association between sulfur dioxide concentration and illness ratio in only one age group could be regarded as a chance happening. Yet the elderly patient with severe chronic bronchitis would appear to be especially susceptible, since this group is also heavily involved in the excess deaths of acute pollution episodes in London and New York. Accordingly, it seems reasonable to infer, as the author did, that there is in all likelihood an effect of high daily levels of sulfur dioxide on older patients with advanced bronchitis; this effect is one of precipitating illness and of exacerbating symptoms. The technique used in this study of developing a personal pollution index estimated for each patient with regard to his place of employment and residence adds confidence to the authors' analysis.

---

* SO_2 values in this study were originally reported in ppm.

An increase in respiratory symptoms was noted among elderly emphysematous individuals in a rest home in the Ruhr area during December 9–13, 1959. The complaints included breathlessness, throat and eye irritation, and depression and apathy (without further specification). The estimated mean indoor sulfur dioxide concentration was 540 $\mu g/m^3$ [0.20 ppm]* for 24 hours. Although the method of measurement was not specified, it probably was the Woesthoff method. For the same period, at an estimated 24-hour indoor sulfur dioxide concentration of 270 $\mu g/m^3$ [0.10 ppm]* for 4 days, there was an increase in functional disturbance.[7, 8, 73]

Patients with preexisting chronic respiratory disease in general seem to have an exacerbation of their symptoms when the sulfur dioxide level exceeds 600 $\mu g/m^3$ (approximately 0.2 ppm) for 24 hours or more in the presence of a substantial amount of unmeasured particulate pollution.

6. Miscellaneous Studies

Although the major action of sulfur oxides has been assumed to be by surface contact on the mucous membranes of the respiratory system, the possibility of effects on the total body system (systemic effects) must be considered. Sulfur dioxide is absorbed into the blood stream and may produce subtle effects on other organs. A few of the studies mentioned previously have suggested that sulfur oxides are a cause of several kinds of illness, rather than respiratory illnesses alone. Even if it is assumed that sulfur oxides are a cause of these results, the effect observed may be secondary to respiratory tract irritation, or indeed, to errors in diagnostic classifications.

Reports from Czechoslovakia[74, 75] suggest that sulfur oxides may have an influence on blood formation in children. However, no details or supporting data for their conclusions are given.

Elfimova *et al.*[76, 77] compared "biochemical blood tests" in areas enduring an average of 2,000 $\mu g/m^3$ (0.70 ppm) SO_2 with an area having an average of 830 $\mu g/m^3$ (0.29 ppm). Sulfur dioxide could be detected in a much larger proportion of blood samples from subjects in the polluted districts than in those from subjects in the clean district. Ascorbic acid levels were also measured and presumably were lower in the polluted area, but the two published versions seem to have conflicting values. At best, these studies indicate a need for further research, probably in the laboratory, and are not yet sufficiently described to warrant their utilization in air quality criteria.

Yanysheva[78] found an excess of respiratory disease, anemia, and rickets among Russian children living in a highly polluted area (13,300 $\mu g/m^3$ to 33,000 $\mu g/m^3$, or 4.6 ppm to 11.5 ppm)* compared to a control area. Epidemiologic methods and bias factors such as social class are not well described, although the types of anemia reported are the same as mentioned above. Sulfur dioxide levels in these three studies are extremely high even in the "clean" areas, and possibly some error has been made in reporting (or analysis of SO_2), although Yanysheva describes symptoms of respiratory disturbances and material damage that would, in fact, indicate very high levels.

D. EFFECTS OF INDUSTRIAL EXPOSURE

Studies of workers occupationally exposed to pollutants provide a unique opportunity to isolate the effects of various chemicals. By carefully choosing industrial situations, populations can be found which have been exposed to rather high levels of specific pollutants for a long period of time. An extremely serious disadvantage of this type of study for the preparation of air quality criteria is that the exposed workers are gainfully employed, and lack of disease in them may not indicate safety of the concentrations to a general population which includes the elderly and infirm. Sensitive people are either not selected for employment, or are soon lost to employment.

---

* The numbers in brackets are the editor's; an equivalency of 2,700 $\mu g/m^3 = 1$ ppm SO_2 was used to assure consistency with any conversions made by the authors.

* SO_2 values in this study were originally reported in mg/m^3.

In conjunction with previously described Berlin, New Hampshire, studies, Ferris *et al.*[79] compared samples of workers from a pulp mill and from an adjacent paper mill. Air concentrations of sulfur dioxide in areas of the pulp mill averaged from 5,720 $\mu g/m^3$ to 37,180 $\mu g/m^3$ (2 ppm to 13 ppm)*. Occupational exposure at the paper mill was almost nonexistent, and most of the workers had been at the same job in each of the plants for a number of years. Some of the men in the pulp mill were exposed to chlorine as well as sulfur dioxide, and some only to sulfur dioxide. No significant differences were found in respiratory symptoms or in simple tests of pulmonary function between workers in the two mills, but men working with chlorine had slightly lowered respiratory function compared to those exposed to sulfur dioxide alone. A unique feature of this industrial report is that cigarette smoking was allowed for in the analysis. The working men of both mills had a lower prevalence of respiratory disease than the male population of Berlin, New Hampshire, suggesting that working populations may not be representative of the general population.

Skalpe[80] also compared workers from pulp and paper industries in Norway. Sulfur dioxide concentrations at the pulp mill varied between 5,720 $\mu g/m^3$ and 102,960 $\mu g/m^3$ (2 ppm to 36 ppm)*. No significant difference in age or smoking habits was found between the groups, but exact comparisons similar to those of Ferris were not made. A significantly higher frequency of cough, expectoration, and shortness of breath on exertion was found in the exposed pulp mill workers, the difference being greater in age groups under 50 years. In these younger workers the maximal expiratory flow rate was also significantly lower, but no difference was found in the older men. The older workers had been employed for a longer time, and possibly those most susceptible had already been eliminated from the working population. Even the younger workers, however, had all been employed for over 1 year.

Anderson[81] compared oil refinery workers in South Persia with similar workers not exposed to sulfur dioxide. Exposure periods ranged from 1 to 19 years, and daily concentrations of sulfur dioxide varied between zero $\mu g/m^3$ and 71,500 $\mu g/m^3$ (25 ppm)* with occasional figures of 286,000 $\mu g/m^3$ (100 ppm) recorded. No differences were found between the two groups in several measures of health, including pulmonary function, chest X-rays, or clinical examination.

Kehoe *et al.*[82] studied 100 refrigeration plant workers who, for periods of 4 to 12 years, had been exposed to concentrations exceeding 28,600 $\mu g/m^3$ (10 ppm)* of sulfur dioxide. Compared to workers in other areas of the plant where no significant exposure to sulfur dioxide was experienced, the exposed workers were found to have a significantly higher incidence of nasal pharyngitis, alterations in sense of smell and taste, and an increased sensitivity to other irritants. In addition, a higher incidence of shortness of breath, increased fatigue, and abnormal reflexes was found in the exposed groups. The incidence of "colds" was not different between the two groups, but the duration of respiratory illnesses was longer in the exposed group. This study was performed shortly after the limitation of exposures by control measures, and concentrations in prior years may have ranged from 228,800 $\mu g/m^3$ to 286,000 $\mu g/m^3$ (80 ppm to 100 ppm). The data presented are insufficient to determine whether the severe effects noted were limited to the workers who had been exposed to the higher concentrations of previous years.

---

* SO_2 values in this study were originally reported in ppm.

Thus the industrial exposures suggest that no significant effect on health or pulmonary function can be noted at exposures up to about 5,700 $\mu g/m^3$ (2 ppm) for the working hours of the day over a period of several years. In these situations there is a lack of a demonstrable effect of exposure to relatively pure sulfur dioxide at concentrations that are well above those implicated in ambient exposures. However, as noted previously, studies of occupational exposure have severely limited value for the preparation of ambient air quality criteria.

The effects on both morbidity and mortality demonstrated for community exposures are probably a result of the synergism of the sulfur dioxide and the suspended particulates found in the ambient atmosphere rather than the effects of either pollutant alone. The data regarding the industrial exposures are summarized in Table 9–3.

E. SUMMARY

This chapter reviews epidemiologic studies of the relationship between pollutant concentrations and their effects on health. Indices varying from disturbance of lung function to death are considered.

In considering levels of pollution at which health effects occur, concentrations are given in the original units employed since conversion from one method to another is not recommended. Attention must also be given to the averaging time employed in the original observation; because of the typical log-normal distribution of sulfur oxide concentrations, long-term averages are considerably lower than short-term averages in the same locations.

Short-term exposure to sulfur oxides may produce symptoms and illness in otherwise healthy people, but the magnitude of the effects and concentrations necessary to produce the effects is not well defined, and exposures are generally substantially higher than the levels cited in the epidemiologic studies.

Over the years, a number of acute air pollution episodes have been reported in the United States and abroad. Both oxides of sulfur and particulate matter have contributed significantly to the health effects associated with these episodes.

In London, a rise in the daily death rate has been detected when the concentrations of sulfur dioxide rose abruptly to levels at or about 715 $\mu g/m^3$ (0.25 ppm) as measured by the hydrogen peroxide titrimetric

Table 9–3.—INDUSTRIAL EXPOSURES

Comparison	Level of Exposure, $\mu g/m^3$ SO_2	Effects	Reference	
			No.	Author
Pulp versus paper mill workers Berlin, New Hampshire	5,700 to 37,000	No difference in pulmonary function or respiratory disease prevalence rates after control for cigarette smoking	79	Ferris *et al.*
Pulp versus paper mill workers Norway	5,700 to 103,000; many over 30,000	Excess cough, sputum, and expectoration under age 50. No effect over age 50.	80	Skalpe
Exposed versus non-exposed oil refinery workers, South Persia	0 to 71,500; over 286,000 occasionally	No effect on pulmonary function, chest X-ray or clinical examination	81	Anderson
Exposed versus non-exposed refrigeration plant workers, United States	over 28,000; 286,000 at times	Increased shortness of breath and fatigue and abnormal reflexes. Longer duration of "colds." No effect on respiratory disease *incidence*.	82	Kehoe *et al.*

method) in the presence of smoke at 750 $\mu g/m^3$. The elderly and patients with heart or lung disease were predominantly affected, but others have also been involved, particularly when pollution reached higher levels. Daily concentrations of sulfur dioxide in excess of 1,500 $\mu g/m^3$ for 1 day (0.52 ppm) in conjunction with levels of suspended particles exceeding 2,000 $\mu g/m^3$ have resulted in an increase in the death rate of 20 percent or more over base line levels.

In New York City, sulfur dioxide concentrations of 1,500 $\mu g/m^3$ (0.52 ppm) (as measured by the hydrogen peroxide titrimetric method) and suspended particulate matter, measured as a soiling index of 6 cohs or greater, have led to increased mortality.

In Rotterdam, a 24-hour mean concentration of 500 $\mu g/m^3$ SO_2 (0.19 ppm) (hydrogen peroxide titrimetric method) lasting for 3 to 4 days has led to an increased mortality rate. This observation is especially significant because particulate levels are very low in Rotterdam. Even lower levels of pollution from 300 $\mu g/m^3$ to 500 $\mu g/m^3$ SO_2 (0.11 ppm to 0.19 ppm) for 24 hours, are thought possibly to increase mortality.

Morbidity rates have been increased in all episodes which have resulted in an increased mortality. Thus, in London, an increase in emergency admissions to hospitals, calls to general practitioners, and certified sickness absence from work has been noted. In Rotterdam increased hospital admissions for respiratory disease, particularly in older persons, has occurred when SO_2 levels have risen to 300 $\mu g/m^3$ to 500 $\mu g/m^3$ (0.11 ppm to 0.19 ppm); there has also been an increase in absenteeism from work which reached 30 percent for all ages and 50 percent to 100 percent in persons age 45 and over.

A survey of emergency clinic visits to major New York City hospitals in November 1953 revealed an increase in visits for upper respiratory infections and cardiac diseases in both children and adults in all of the four hospitals studied. Sulfur dioxide levels ranged between 200 $\mu g/m^3$ and 2,460 $\mu g/m^3$ (0.07 ppm to 0.86 ppm) during the period from November 12 to 24, and hospital admissions had clearly increased by November 16, at which time concentrations had not yet exceeded 715 $\mu g/m^3$ (0.25 ppm); smoke shade at this time was close to 3 coh units.

The most striking effects have been observed in association with exceptional episodes of air pollution. However, lower and more persistent concentrations are also correlated with mortality and morbidity. British studies have indicated an association of bronchitis death rates with air pollution concentrations that is apparently independent of social class differences, but which also do not specify the role played by sulfur oxides. Instead, these studies treat of a combination of sulfur oxides and particulates. In one major study of Eston, sulfur dioxide values for a year averaged 115 $\mu g/m^3$ (0.040 ppm) in the dirty area and 74 $\mu g/m^3$ (0.026 ppm) in the cleaner area. Smoke values were 160 $\mu g/m^3$ and 80 $\mu g/m^3$ respectively.

In the United States, the Buffalo study indicated an effect on chronic bronchitis disease mortality for men aged 50 to 59 in the low social classes. This finding of air pollution exerting a special effect on the low social classes appears to be consistent with the British findings.

A study in Genoa, Italy, made use of housewives. One of the groups surveyed included women all 65 or more years of age who were nonsmokers, who had lived for a long period in the same area and who had no industrial exposure.

There was a finding of increased frequency of cough, sputum, dyspnea, and bronchitis in the moderate-pollution area over the cleanest area. A much sharper difference in frequency between the cleanest and the dirtiest area was found. The cleanest area had an annual mean of 80 $\mu g/m^3$ (0.028 ppm) of sulfur dioxide, the mean of the moderately clean area was 105 $\mu g/m^3$ (0.037 ppm), and the dirty area averaged 265 $\mu g/m^3$ (0.093 ppm). The measurement of sulfur dioxide was recorded by a technique analogous to the volumetric method of the British.

This study also showed a correlation between the frequency of bronchitis and the annual mean of sulfur dioxide levels for the seven districts of Genoa whereas the correlation between the frequency of bronchitis and suspended matter and dustfall was not statistically significant.

In Berlin, New Hampshire, no effect was observed for long-term exposure in areas with sulfation levels averaging 610 $\mu g/100$ cm^2-day. The authors recognized that the lack of results may have been influenced by the narrow differences in air quality between the areas compared, and they also noted that there probably had been a selected migration of diseased persons to the less polluted areas, which could have affected the results.

Another morbidity study which included housewives was that conducted in Nashville. This showed a direct correlation between illnesses for all causes for housekeeping white females, 15 to 64 years of age, and sulfur dioxide levels. It also showed that morbidity from cardiovascular disease in the 55 and older age group, for both sexes, was twice as high in the most polluted area as compared with the least. However, cigarette smoking was not taken into account and the adjustment made for socioeconomic status may not have been adequate.

The study of Port Kembla, Australia, had shown differences in respiratory symptoms for areas which had long-term values of 95 $\mu g/m^3$ SO_2 (0.034 ppm) and 25 $\mu g/m^3$ (0.009 ppm) measured by absorption in a sulfuric acid solution containing hydrogen peroxide. The study is unusual in that the source of pollution was the single stack of a smelter and daily averages were up to 17 times the annual mean. However, the findings that respiratory symptoms disappeared when pollution was reduced through installation of a higher stack, would appear to confirm an effect of sulfur dioxide at the concentrations originally measured, provided that no other change took place which could account for the disappearance of symptoms.

The study of school children in Great Britain indicated that they experienced increased frequency and severity of respiratory diseases when the long-term levels exceeded about 120 $\mu g/m^3$ (0.046 ppm) SO_2 and 100 $\mu g/m^3$ for smoke.

Several studies have been noted relating daily variations in air pollution with changes in the clinical condition of patients with chronic lung disease. A good deal of information exists on the effects of exposure to moderately elevated levels of SO_2 lasting a day to 3 or 4 days. A daily average level of about 600 $\mu g/m^3$ of SO_2 (0.21 ppm) has caused accentuation of symptoms in persons with chronic respiratory disease on the day following the high SO_2 level if particulate matter at a similar concentration was also present.

This finding by Lawther was also observed by Carnow in Chicago. He too noticed a sharp rise in illness rates on the day following exposure to 715 $\mu g/m^3$ (0.25 ppm) of sulfur dioxide (West-Gaeke method) or more for patients 55 and over with severe bronchitis. Particulate matter was also present in the atmosphere.

The study of an elderly emphysematous population in a rest home in the Ruhr area of Germany demonstrated that complaints including breathlessness, throat and eye irritation, and "depression and apathy without further specification" increased at estimated indoor sulfur dioxide concentrations of 540 $\mu g/m^3$ (0.20 ppm) for 24 hours. There was an increase in functional disturbance at an average daily level of 270 $\mu g/m^3$ (0.10 ppm) for 4 days, again an estimated indoor level, measurement presumably being made by the Woesthoff method.

The analyses of the numerous epidemiological studies discussed clearly indicate an association between air pollution, as measured by particulate matter accompanied by sulfur dioxide, and health effects of varying severity. This association is most firm for the short-term air pollution episodes.

There are probably no communities which do not contain a reservoir of individuals with impaired health who are prime targets for the effects of elevated levels of particulate matter and sulfur oxides. However, to show small changes in deaths associated with coincident higher levels of air pollutants require extremely large populations. In small cities, these small changes cannot be detected statistically.

The epidemiologic studies concerned with increased mortality also show increased morbidity. Again, increases in morbidity as measured, for example, by increases in hospital admissions or emergency clinic visits, are most easily detected in major urban areas.

It is believed that, for the large urban communities which are routinely exposed to relatively high levels of pollution, sound statistical analysis can detect with confidence the small changes in daily mortality which are associated with functions in pollution concentrations. Unfortunately, only limited analysis has thus far been made, and this has been attempted only in London and in New York.

The association between longer-term community exposures to particulate matter for respiratory disease incidence and prevalence rates is conservatively believed to be intermediate in its reliability. Because of the reenforcing nature of the studies conducted to date, the conclusions to be drawn from this type of study can be characterized as probable.

The association between long-term residence in a polluted area and chronic disease morbidity and mortality is somewhat more conjectural. However, in the absence of other explanations, the findings of increased morbidity and of increased death rates for selected causes, independent of economic status, must still be considered consequential.

F. REFERENCES

1. Hill, A. B. "The Environment and Disease: Association or Causation." Proc. Royal Soc. Med., Vol. 58, pp. 295–300, 1965.
2. Cassell, E. G., Walter, E. W., Mountain, J. D., Diamond, J. R., Mountain, I. M., and McCarroll, J. R. "Reconsiderations of Mortality as a Useful Index of the Relationship of Environmental Factors to Health." Am. J. Pub. Health, Vol. 58, pp. 1653–1657, 1968.
3. Firket, J. "Fog Along the Meuse Valley." Trans. Faraday Soc., Vol. 32, pp. 1102–1197, 1936.
4. Firket, J. "The Cause of the Symptoms Found in the Meuse Valley During the Fog of December 1930." Bull. Roy. Acad. Med. Belgium, Vol. 11, pp. 683–739, 1931.
5. Schrenk, H. H., Heimann, H., Clayton, G. O., Gafafer, W. M., Wexler, H. "Air Pollution in Donora, Pennsylvania. Epidemiology of the Unusual Smog Episode of October 1948." Pub. Health Bull. No. 306, Fed. Sec. Agency, Washington, D.C., 1949.
6. Wilkins, E. T. "Air Pollution Aspects of the London Fog of December 1952." Roy. Meteorol. Soc. J., Vol. 80, pp. 267–271, 1954.
7. Brasser, L. J., Joosting, P. E., and von Zuilen, D. "Sulfur Dioxide—To What Level is it Acceptable?" Research Institute for Public Health Engineering, Delft, Netherlands, Report G–300, July 1967. (Originally published in Dutch, September 1966.)
8. Joosting, P. E. 'Air Pollution Permissibility Standards Approached from the Hygienic Viewpoint." Ingenieur, *79* (50) :A739–A747, 1967.
9. Anderson, D. O. "The Effects of Air Contamination of Health, Part I." Canad. Med. Assoc. J., Vol. 97, pp. 528–536, 1967.
10. Gore, A. T. and Shaddick, C. W. "Atmospheric Pollution and Mortality in the County of London." Brit. J. Prev. Soc. Med., Vol. 12, pp. 104–113, 1958.
11. Burgess, S. G. and Shaddick, C. W. "Bronchitis and Air Pollution." Roy. Soc. Hlth. J., Vol. 79, pp. 10–24, 1959.
12. Scott, J. A. "The London Fog of December 1962." Medical Officer, Vol. 109, pp. 250–252, 1963.
13. Martin, A. E. and Bradley, W. "Mortality, Fog and Atmospheric Pollution." Monthly Bull. Ministry of Health, Vol. 19, pp. 56–59, 1960.
14. Martin, A. E. "Mortality and Morbidity Statistics and Air Pollution." Proc. Roy. Soc. Med., Vol. 7, pp. 969–975, 1964.
15. Lawther, P. J. "Compliance with the Clean Air Act: Medical Aspects." J. Inst. Fuels (London), Vol. 36, pp. 341–344, 1963.
16. Greenburg, L., Jacobs, M. B., Drolette, B. M., Field, F., and Braverman, M. M. "Report of an Air Pollution Incident in New York City, November 1953." Pub. Health Reports, Vol. 77, pp. 7–16, 1962.
17. Greenburg, L., Erhardt, C., Field, F., Reed, J. I., and Seriff, N. S. "Intermittent Air Pollution Episodes in New York City, 1962." Pub. Health Rpts., 78, pp. 1061–1065, 1963.
18. McCarroll, J. and Bradley, W. "Excess Mortality as an Indicator of Health Effects of Air Pollution." Am. J. Public Health, Vol. 56, pp. 1933–1942, 1966.
19. Glasser, M., Greenburg, L., and Field, F. "Mortality and Morbidity During a Period of High Levels of Air Pollution, New York, November 23-25, 1966." Arch. Environ. Health, Vol. 15, pp. 684–694, 1967.
20. Leonard, A. G., Crowley, D., and Belton, J. Atmospheric pollution in Dublin during the years 1944 to 1950. Royal Dublin Soc. Sci. Proc. 25:166–167, 1950.
21. "Report of the International Joint Commission, United States and Canada, on the Pollution of the Atmosphere in the Detroit River Area." International Joint Commission (United States and Canada), Washington/Ottawa, 1960, 115 pp.
22. Watanabe, H. "Air Pollution and its Health Effects in Osaka, Japan." Preprint. (Presented at the 58th Annual Meeting of the Air Pollution Control Association, Toronto, Canada, June 20–24, 1965.)

23. Greenburg, L., Field, F., Reed, J. I., and Erhardt, C. L. "Air Pollution and Morbidity in New York City." J. Amer. Med. Assoc., Vol. 182, pp. 161–164, 1962.
24. McCarroll, J. R., Cassell, E. G., Walter, E. W., Mountain, J. D., Diamond, J. R., and Mountain, I. R. "Health and the Urban Environment. V. Air Pollution and Illness in a Normal Urban Population." Arch. Environ. Health, Vol. 14, pp. 178–184, 1967.
25. "Atmospheric Pollutants." World Health Organization, Technical Report Series 271, Geneva, 1964.
26. Kurland, Leonard T. Personal communication, Nov. 1, 1968.
27. Sterling, T. D., Phair, J. J., Pollack, S. V., Schumsky, D. A., and deGrott, I. "Urban Morbidity and Air Pollution." Arch. Environ. Health, Vol. 13, pp. 158–170, 1966.
28. Markush, R. E. "National Chronic Respiratory Disease Mortality Study. I. Prevalence and Severity at Death of Chronic Respiratory Disease in the United States, 1963." J. Chron. Dis., Vol. 21, pp. 129–141, 1968.
29. Buck, S. F. and Brown, D. A. "Mortality from Lung Cancer and Bronchitis in Relation to Smoke and Sulfur Dioxide Concentration, Population Density, and Social Index." Tobacco Research Council, London, Research Paper 7, 1964.
30. Pemberton, J. and Goldberg, C. "Air Pollution and Bronchitis." Brit. Med. J., Vol. 2, pp. 567–570, 1954.
31. Gorham, E. "Pneumonia and Atmospheric Sulphate Deposit." Lancet, Vol. 2, pp. 287–288, 1959.
32. Fairbairn, A. S. and Reid, D. D. "Air Pollution and Other Local Factors in Respiratory Disease." Brit. J. Prev. Soc. Med., Vol. 12, pp. 94–103, 1958.
33. Daly, C. "Air Pollution and Causes of Death." Brit. J. Prev. Soc. Med., Vol. 13, pp. 14–27, 1959.
34. Wicken, A. J. and Buck, S. F. "Report on a Study of Environmental Factors Associated with Lung Cancer and Bronchitis Mortality in Areas of North East England." Tobacco Research Council, London, Research Paper 8, 1964.
35. Burn, J. L. and Pemberton, J. "Air Pollution, Bronchitis, and Lung Cancer in Salford." Int. J. Air and Water Pollution, Vol. 7, p. 5, 1963.
36. Winkelstein, W., Kantor, S., Davis, E. W., Maneri, C. S., and Mosher, W. E. "The Relationship of Air Pollution and Economic Status to Total Mortality and Selected Respiratory System Mortality in Man." Arch. Environ. Health, Vol. 14, pp. 162–169, 1967.
37. Winkelstein, W., Kantor, S., Davis, E. W., Maneri, C. S., Mosher, W. E. "The Relationship of Air Pollution and Economic Status to Total Mortality and Selected Respiratory System Mortality in Man (II. Oxides of Sulfur)." Arch. Environ. Health, Vol. 15, pp. 401–405, 1968.
38. Winkelstein, W. "The Relationship of Air Pollution to Cancer of the Stomach." (Presented at the Air Pollution Medical Research Conference, Denver, Colorado, July 22–24, 1968.)
39. Reid, Donald D. Personal communication, Nov. 28, 1968.
40. Zeidberg, L. D. Prindle, R. A., and Landau, E. "The Nashville Air Pollution Study. III. Morbidity in Relation to Air Pollution." Am. J. Pub. Health, Vol. 54, pp. 85–97, 1964.
41. Zeidberg, L. D., Horton, R. J. M., and Landau, E. "The Nashville Air Pollution Study. V. Mortality from Diseases of the Respiratory System in Relation to Air Pollution." Arch. Environ. Health, Vol. 15, pp. 214–224, 1967.
42. Sprague, H. A. and Hagstrom, R. M. "The Nashville Air Pollution Study: Mortality Multiple Regression." (Presented at the Air Pollution Medical Research Conf., Denver, Colorado, July 22–24, 1968.) Arch. Environ. Health, Vol. 18, pp. 503–507, 1969.
43. Anderson, D. O. "The Effects of Air Contamination on Health: Part III." Canadian Med. Assoc. J., Vol. 97, pp. 802–806, Sept. 23, 1967.
44. Petrilli, R. L., Agnese, G., and Kanitz, S. "Epidemiology Studies of Air Pollution Effects in Genoa, Italy." Arch. Environ. Health, Vol. 12, pp. 733–740, 1966.
45. Toyama, T. "Air Pollution and its Health Effects in Japan." Arch. Environ. Health, Vol. 8, pp. 153–173, 1964.
46. Bell, A. "The Effects on the Health of Residents of East Port Kembla. Part II." In: Air Pollution by Metallurgical Industries. Div. Occup. Health, New South Wales Dept. of Pub. Health, Sydney, Australia, Vol. 2, 1962, pp. 1–144.
47. Goldsmith, J. R. "Effects of Air Pollution on Human Health." Academic Press, 1967.
48. Holland, W. W. and Stone, R. W. "Respiratory Disorders in United States East Coast Telephone Men." Amer. J. of Epidemiology, Vol. 82, p. 92, 1965.
49. Holland, W. W., Reid, D. D., Seltser, R., and Stone, R. W. "Respiratory Disease in England and the United States. Studies of Comparative Prevalence." Arch. Environ. Health, Vol. 10, pp. 338–345, 1965.
50. Holland, W. W. and Reid, D. D. "The Urban Factor in Chronic Bronchitis." Lancet, Vol. 1, pp. 445–448, 1965.
51. Bates, D. V. "Air Pollution and Chronic Bronchitis." Arch. Environ. Health, Vol. 14, pp. 220–224, 1967.
52. Ferris, B. G., Jr. and Anderson, D. O. "The Prevalence of Chronic Respiratory Disease in a New Hampshire Town." Am. Rev. Resp. Dis., Vol. 86, pp. 165–177, 1962.
53. Anderson, D. O., Ferris, B. G., and Zickmantel, R. "Levels of Air Pollution and Respiratory Disease in Berlin, New Hampshire." Am. Rev. Resp. Dis., Vol. 90, pp. 877–887, 1964.

54. Anderson, D. O. and Ferris, B. G. "Air Pollution Levels and Chronic Respiratory Disease." Arch. Environ. Health, Vol. 10, pp. 307–311, 1965.

55. Kenline, P. A. "In Quest of Clean Air in Berlin, New Hampshire." U.S. Dept. of Health, Education, and Welfare, Public Health Service, Division of Air Pollution, 1962.

56. Prindle, R. A., Wright, G. W., McCaldin, R. O., Marcus, S. C., Lloyd, T. C., and Bye, W. E. "Comparison of Pulmonary Function and Other Parameters in Two Communities with Widely Different Air Pollution Levels." Am. J. Pub. Health, Vol. 53, pp. 200–218, 1963.

57. Dohan, F. C. "Air Pollutants and Incidence of Respiratory Disease." Arch. Environ. Health, Vol. 3, pp. 387–395, Oct. 1961.

58. Dohan, F. C. and Taylor, E. W. "Air Pollution and Respiratory Disease, a Preliminary Report." Am. J. Med. Sci., Vol. 240, pp. 337–339, 1960.

59. "Report on an Enquiry into the Incidence of Incapacity for Work: Part II. Incidence of Incapacity for Work in Different Areas and Occupations." Ministry of Pensions and National Insurance, Her Majesty's Stationery Office, London, 1965.

60. Verma, M. P., Schilling, F. J., and Becker, W. H. "Epidemiological Study of Illness Absences in Relation to Air Pollution." (Presented at the Air Pollution Medical Research Conf., Denver, Colorado, July 22–24, 1968.) Arch. Environ. Health, Vol. 18, pp. 536–543, 1969.

61. Reid, D. D. "Air Pollution and Respiratory Disease in Children." Second International Symposium on Bronchitis, The Netherlands, 1964.

62. Douglas, J. W. B. and Waller, R. E. "Air Pollution and Respiratory Infection in Children." Brit. J. Prev. Soc. Med., Vol. 20, pp. 1–8, 1966.

63. Holland, W. W. and Elliott, A. "Cigarette Smoking, Respiratory Symptoms, and Antismoking Propaganda: An Experiment." Lancet, Vol. 1, pp. 41–43, 1968.

64. Lunn, J. E., Knowelden, J., and Handyside, A. J. "Patterns of Respiratory Illness in Sheffield Infant School Children." Brit. J. Prev. Soc. Med., Vol. 21, pp. 7–16, 1967.

65. Manzhenko, E. G. "The Effect of Atmospheric Pollution on the Health of Children." Hyg. and Sanit. (English translation), Vol. 31, pp. 126–128, 1966.

66. Spicer, W. S., Jr., Storey, P. B., Morgan, W. K. C., Kerr, H. P., and Standiford, N. E. "Variation in Respiratory Function in Selected Patients and its Relation to Air Pollution." Am. Rev. Resp. Dis., Vol. 86, pp. 705–712, 1962.

67. Shephard, R. J., Turner, M. E., Carey, G. C. R., and Phair, J. J. "Correlation of Pulmonary Function and Domestic Microenvironment." J. Appl. Physiol., Vol. 15, pp. 70–76, 1960.

68. Lawther, P. J. "Climate, Air Pollution, and Chronic Bronchitis." Proc. Roy. Soc. Med., Vol. 51, pp. 262–264, 1958.

69. Waller, R. E. and Lawther, P. J. "Some Observations on London Fog." Brit. Med. J., p. 1356, Dec. 3, 1955.

70. Angel, J. H., Fletcher, C. M., Hill, J. D., and Tinker, C. M. "Respiratory Illness in Factory and Office Workers." Brit. J. Dis. Chest, Vol. 59, pp. 66–79, 1965.

71. Carnow, B. W., Lepper, M. H., Shekelle, R. B., and Stamler, J. "The Chicago Air Pollution Study: Acute Illness and SO_2 Levels in Patients with Chronic Bronchopulmonary Disease." (Presented at National Air Pollution Control Association Meeting, Minneapolis, June 24, 1968.)

72. "Air Quality Data (1964–1965)." U.S. Dept. of Health, Education, and Welfare, Public Health Service, 1966.

73. Joosting, P. E. Personal communication, Dec. 16, 1968.

74. Kapalin, V. "The Red Blood Picture in Children from Different Environments." Rev. Czech. Med., Vol. 9, pp. 65–87, 1963.

75. Petr, B. and Schmidt, P. "The Influence of an Atmosphere Contaminated with Sulfur Dioxide and Nitrous Gases on the Health of Children." Zeit. Gesamt. Hyg. Grenzgeb., Vol. 13, pp. 34–48, Jan. 1967.

76. Elfimova, E. V. and Shashkov, V. S. "The Effect of Sulfur Dioxide Gas in the Atmospheric Air on Some Biochemical Indexes of the Blood in Man." Gig. i Sanit., Vol. 25, pp. 18–22, 1960.

77. Elfimova, E. V. and Pushkina, N. N. "Ascorbic Acid Excretion in the Urine as an Indicator of the Harmful Effects of Atmospheric Pollution." Hyg. Sanit. (English translation), Vol. 31, pp. 255–257, 1966.

78. Yanysheva, N. Ya. "The Effect of Atmospheric Air Pollution by Discharges from Electric Power Plants and Chemical Combines on the Health of Nearby Inhabitants." Gig. i Sanit., Vol. 8, pp. 15–20, 1957. In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases, Translated by B. S. Levine, Vol. 1, pp. 98–104, 1960.

79. Ferris, B. J., Burgess, W. A., and Worcester, J. "Prevalence of Chronic Respiratory Disease in a Pulp Mill and a Paper Mill in the United States." Brit. J. Ind. Med., Vol. 24, pp. 26–37, 1967.

80. Skalpe, T. O. "Long-Term Effects of Sulphur Dioxide Exposures in Pulp Mills." Brit. J. Ind. Med., Vol. 21, pp. 69–73, 1964.

81. Anderson, A. "Possible Long-Term Effects of Exposure to Sulfur Dioxide." Brit. J. Ind. Med., Vol. 7, pp. 82–86, 1950.

82. Kehoe, R. A., Machle, W. F., Kitzmiller, K., and LeBlanc, T. J. "On the Effects of Prolonged Exposure to Sulphur Dioxide." J. Ind. Hyg., Vol. 14, pp. 159–173, 1932.

BLANK PAGE

Chapter 10

SUMMARY AND CONCLUSIONS

Table of Contents

Page

A. SUMMARY 153
1. General 153
2. Relationship of Maximum Concentrations to Average Concentrations 154
3. Effects on Health 155
4. Effects on Visibility 158
5. Effects on Materials 159
6. Effects on Vegetation 160

B. CONCLUSIONS 161
1. Effects on Health 161
2. Effects on Visibility 162
3. Effects on Materials 162
4. Effects on Vegetation 162

C. RESUME 162

Chapter 10

SUMMARY AND CONCLUSIONS

A. SUMMARY

1. General

This document presents criteria of air quality in terms of the effects empirically obtained and published for various concentrations of one family of pollutants, the sulfur oxides, their acids and acid salts. These effects do not, for the most part, derive solely from the presence of sulfur oxides in the atmosphere. They are the effects that have been observed when various concentrations of sulfur oxides, along with other pollutants, have been present in the atmosphere. Many of these effects are produced by a combination of sulfur oxides pollution and undifferentiated particulate matter; the contributions of each class are difficult to distinguish. Moreover, laboratory studies have shown that a combination of sulfur oxides and particulates may produce an effect that is greater than the sum of the effects caused by these pollutant classes individually. Because of the interactions between pollutants, and the reactions of pollutants with oxygen and with water in the atmosphere, and because of the influence of sunlight and temperature on these reactions, the criteria for sulfur oxides can not be presented as exact expressions of cause and effect that have been replicated from laboratory to laboratory. They are presented as useful statements of the effects that can be predicted when sulfur oxides are present in the atmosphere; they are derived from a careful evaluation of what has so far been reported.

The sulfur oxides are common atmospheric pollutants which arise mainly from the combustion of fuels. Solid and liquid fossil fuels contain sulfur, usually in the form of inorganic sulfides or sulfur-containing organic compounds. Combustion of the fuel forms about 25 to 30 parts of sulfur dioxide to 1 part of sulfur trioxide.

Sulfur dioxide is a non-flammable, nonexplosive colorless gas that most people can taste at concentrations from 0.3 ppm to 1 ppm (about 0.9 mg/m^3 to 3 mg/m^3) in air. At concentrations above 3 ppm (about 8.6 mg/m^3), the gas has a pungent, irritating odor. In the atmosphere, sulfur dioxide is partly converted to sulfur trioxide or to sulfuric acid and its salts by photochemical or catalytic processes. Sulfur trioxide is immediately converted to sulfuric acid in the presence of moisture. The degree of oxidation of sulfur dioxide in the atmosphere is dependent on a number of factors, including residence time, amount of moisture present, and the intensity and duration of sunlight and its spectral distribution. The amounts of catalytic material, hydrocarbons and nitrogen oxides, and the amounts of sorptive and alkaline materials present, also affect the oxidation process.

In the United States, sulfur dioxide is most commonly measured by the colorimetric West-Gaeke (pararosaniline) and the conductometric methods. The West-Gaeke method is specific for sulfur dioxide and sulfite salts. The method has been modified to compensate for interferences produced by the presence of nitrogen oxides, ozone, or heavy metal salts in the sample, and the modified method is the method of choice of the National Air Pollution Control Administration. Conductometric methods measure sulfur dioxide concentrations as a function of change in the electroconductivity of a solution. These methods are general, in that they react to changes in electroconductivity brought on by other soluble gases, as well as by sulfur dioxide, and the indicated values for sulfur are sometimes very approximate.

The technique most frequently used in Europe to measure sulfur dioxide is the hydrogen peroxide acid titration method, or an automated conductometric version of the same technique. The presence of other acidic or alkaline gases in the sample may affect the results.

The lead peroxide candle is a widely used technique which determines a "sulfation rate." The method gives integrated values for relatively long periods, but provides no indication of short-term fluctuations. It provides only a rough indication of sulfur dioxide concentrations.

Recently, two long-path spectroscopic techniques have been introduced that sense sulfur dioxide concentrations remotely. Although these techniques are complex and expensive, they may eventually be developed to provide a sulfur dioxide "pollution contour" for large areas of a city, as well as pollution concentrations at different elevations.

Sulfuric acid aerosol in suspended particulate material may be measured by titration or by controlled decomposition to sulfur dioxide. The sulfur dioxide can be measured by a number of methods, including spectrophotometry, coulometry, and flame photometry. Particulate sulfate may be analyzed by spectrophotometric or turbidimetric methods.

Each method of measuring sulfur oxides pollution is unique in terms of measuring time resolution, operating costs and skills, time required for analysis, and the specificity of the technique. A single program may make use of both general and specific methods. In selecting the methods to be used in a sampling program, it is especially important to consider the degree to which data obtained from one method can be compared to data obtained from another.

An estimated 28.6 million tons of sulfur dioxide were emitted to the atmosphere of the United States in 1966, as compared with an estimated 23.4 million tons emitted in 1963. The principal share, 58.2 percent, of the 1966 tonnage came from the combustion of coal, primarily for the generation of electric power and for space heating. The combustion of residual fuel oil and other petroleum products, also primarily for power generation and space heating, accounted for 19.6 percent of the total, while the remainder came from the refining of petroleum (5.5 percent), the smelting of sulfur-containing ores (12.2 percent), the manufacturing of sulfuric acid (1.9 percent), the burning of refuse (0.4 percent), and the burning of coal refuse banks (0.4 percent). Paper-making and other industrial operations also contributed minor amounts to the total.

The National Air Pollution Control Administration operates two nationwide programs for surveying sulfur oxides pollution levels in the United States. The National Air Surveillance Network (NASN) takes 24-hour samples of sulfur dioxide from about 100 sites 26 times a year, and the Continuous Air Monitoring Project (CAMP) records 5-minute average concentrations of sulfur dioxide continuously in six large cities—Washington, Philadelphia, Cincinnati, Chicago, St. Louis, and Denver. The NASN program employs the colorimetric, West-Gaeke method of analysis, while the CAMP program uses the electroconductivity technique. Recently, continuous, colorimetric West-Gaeke monitoring devices were installed at the six CAMP locations.

Levels recorded in the CAMP cities over a 6-year period show mean annual concentrations ranging from 0.01 ppm, in San Francisco, to 0.18 ppm, in Chicago, with the averages exceeding, for 1 percent of the time, a concentration between 0.09 ppm and 0.68 ppm. The NASN annual average concentrations ranged from 0.002 ppm, in Kansas City, Missouri, to 0.17 ppm, in New York City. The highest 24-hour average concentration was 0.38 ppm, also in New York City, while the lowest 24-hour averages were below the minimum detectable range of the instruments—below approximately 0.001 ppm. Geographically, the highest values were recorded in the northeastern part of the United States, especially east of the Mississippi River and north of the Ohio River, where large quantities of sulfur-bearing fossil fuels are burned.

2. Relationship of Maximum Concentrations to Average Concentrations

Although it is convenient to discuss the various effects of sulfur dioxide in connection with the average concentrations of the gas over a long period, such as a year, some effects are thought to be associated with the peak concentrations that may occur during the period and to better define the kind and extent of effects that may be occurring in a given community, it is useful to know what these peak values may be.

For a given averaging time, measurements of sulfur dioxide concentrations follow a log-normal frequency distribution. The two statistical parameters used to describe this distribution are the geometric mean and the standard geometric deviation, which is an index of the deviation of the samples from the mean.

Once sufficient sampling data have been gathered, the geometric mean and the standard geometric deviation can be used to calculate the expected maximum concentration, the minimum concentration, and the concentration at any percentile, provided that the averaging time is 1 hour or greater. Further, it is possible to calculate expected maxima for one averaging time from data obtained at another averaging time.

For a given typical, multiple source urban area, a fairly good approximation of the frequency distribution of hourly average concentrations for a year at a given station can be obtained from measurements taken for 24-hour periods on about 26 randomly selected days. The accuracy of the approximation will depend on the number of samples taken, compared to the total number of samples that could have been taken if air sampling had been continuous.

The CAMP data for various years in the period 1962 to 1967 appear to be fairly representative of the distribution of sulfur dioxide concentrations for large U.S. metropolitan areas. The range of standard geometric deviations for the CAMP cities is roughly from 2 to 2.5. These values correspond to hourly maxima that range from 10 to 20 times the annual mean, respectively. Similarly, the 8-hour maximum ranges from about 6 to 10 times the annual mean, and the 1-day maximum is between 4 and 7 times the annual mean.

The ratio of the maximum sulfur dioxide concentration to the average values may be greater for measurements made near a single point source than for a city as a whole. For averaging periods from 4 to 54 minutes, for example, the maximum concentrations encountered near a point source were, respectively, 30 to 160 times the 6-month average value.

3. Effects on Health

The current scientific literature indicates that, for the most part, the effects of the oxides of sulfur on health are related to irritation of the respiratory system. Such injury may be temporary or permanent.

Laboratory studies show that sulfur dioxide can produce bronchoconstriction in experimental animals such as the guinea pig, the dog, and the cat. Dose-response curves have been established for the guinea pig, the most susceptible laboratory animal studied to date. They relate the concentration of sulfur dioxide to the observed increase in pulmonary flow resistance produced by 1-hour exposures. Slight increases in resistance are detectable at 0.16 ppm (460 $\mu g/m^3$) and the changes are readily reversible.

Sulfuric acid and some, but not all, particulate sulfates also produce bronchoconstriction in the guinea pig. The response is highly dependent on particle size, with the smallest particles showing the greatest irritant potency. In this animal, as in man, sulfuric acid and irritant particulate sulfates have a greater irritant potency at a given concentration than does sulfur dioxide alone.

The potentiation by particulate matter of toxic responses to sulfur dioxide (synergism) has been observed under conditions which would promote the conversion of sulfur dioxide to sulfuric acid. The degree of potentiation is related to the concentration of particulate matter. A threefold to fourfold potentiation of the irritant response to sulfur dioxide is observed in the presence of particulate matter capable of oxidizing sulfur dioxide to sulfuric acid. Aerosols of soluble salts of ferrous iron, manganese, and vanadium have been observed to produce this potentiation, although the concentrations used (0.7 mg/m^3 to 1.0 mg/m^3) were considerably greater than any levels of the metals reported in urban air.

Generally speaking, the laboratory work

that has been performed to date with animals has only partial relevance for air quality criteria. In most of the studies, the laboratory environment has not simulated very closely the actual environment. Exposures have been to high and constant concentrations, rather than to the low and fluctuating levels commonly found in the atmosphere. Other normally occurring stresses, such as fluctuating temperature, have not, in general, been applied. These studies do, however, provide valuable information on some of the bioenvironmental relationships that may be involved in the effects of the sulfur oxides on health. The data they provide on synergistic effects show very clearly that information derived from single substance exposures should be applied to ambient air situations only with great caution.

The response of bronchoconstriction in man may be assessed in terms of a slight increase in airway resistance. Normal individuals, exposed to sulfur dioxide via the mouth, exhibit small changes in airway resistance, which are often insufficient to produce any respiratory symptoms. The effects may be even smaller when the subject breathes through his nose. As in animals, sulfuric acid is a much more potent irritant in man than is sulfur dioxide. Again, the irritant effect is highly dependent on particle size.

Laboratory observations of respiratory irritations suggest that most individuals will show a response to sulfur dioxide when exposed for 30 minutes to concentrations of 5 ppm (about 14 mg/m^3) and above. Exposure of certain sensitive individuals to 1 ppm (about 3 mg/m^3) can produce detectable changes in pulmonary function. Similar exposure of these same individuals has, in some instances, produced severe bronchospasm. In most of the studies discussed, an increase in pulmonary flow resistance was the indicator of response employed.

Epidemiologic studies do not have the precision of laboratory studies, but they have the advantage of being carried out under ambient air conditions. In most epidemiologic studies, indices of air pollution level are obtained by measuring selected pollutants, most commonly particulates and sulfur compounds. To use these same studies to establish criteria for individual pollutants is justified by the experimental data on interaction of pollutants. However, in reviewing the results of epidemiologic investigations it should always be remembered that the specific pollutant under discussion is being used as an index of pollution, not as a physicochemical entity.

It has been suggested that industrial experience with sulfur oxides exposures may be relevant to ambient air quality criteria. In the absence of epidemiologic evidence, one might, as a rough approximation, select some fraction of the concentrations reported for industrial exposures. In selecting such a fraction, several factors should be taken into consideration. Industrial exposure, for example, is not continuous, and it may not include the synergistic effects which result from the presence of more than one class of pollutant. Further, the exposed population may not include the segments most susceptible to the effects.

From the epidemiologic studies available, it is easy to conclude that there is an effect of the oxides of sulfur in the ambient atmosphere on the health of the population, and that the degree of effect is related to the degree of pollution. Episodes of acute elevation of oxides of sulfur and other pollutant concentrations have been associated with a larger number of deaths than expected. Those predominantly affected were individuals with chronic pulmonary disease or cardiac disorders, or very young or old individuals. However, the general population has also been involved.

Studies of episodes occurring in London suggest that a rise in the daily death rate occurred when the concentrations of sulfur dioxide rose abruptly to levels at or about 715 μg/m^3 (0.25 ppm) (as measured by the hydrogen peroxide titrimetric method) in the presence of smoke at 750 μg/m^3. A more distinct rise in deaths has been noted generally when sulfur dioxide exceeded 1000 μg/m^3 (0.35 ppm) for one day, and particulate matter reached about 1200 μg/m^3 (measured by the British reflectometer method). Daily concentrations of sulfur dioxide in excess of 1500 μg/m^3 for one day (0.52 ppm) in conjunction with levels of suspended particles exceeding 2000 μg/m^3 appear to have been associated with an increase in the death rate of 20 percent or more over base line levels. This same effect has been observed at lower sulfur dioxide levels when the maximum pollution levels lasted for a longer period.

Air pollution episodes in New York City have been associated with exposures similar to those of the London episodes. In one case, for example, excess deaths were detected in New York following a 24-hour period during which sulfur dioxide concentrations exceeded 1500 μg/m^3 (>0.5 ppm) (as measured by the hydrogen peroxide titrimetric method) and suspended particulate matter was measured as a soiling index of 6 cohs or greater.

For Rotterdam, there have been indications of a positive association between total mortality and exposure for a few days to 24-hour mean concentrations of 500 μg/m^3 (0.19 ppm) sulfur dioxide. Further, it has been reported that: "There is a faint indication that this will happen somewhere between 300 and 500 μg SO_2 per m^3 per 24 hours" (0.11 ppm and 0.19 ppm).

A survey of emergency clinics at major New York City hospitals revealed a rise in visits for upper respiratory infections and cardiac diseases in both children and adults in all 4 hospital studies during a 10-day period of elevated pollution levels. Sulfur dioxide ranged between 200 μg/m^3 and 2460 μg/m^3 (0.07 ppm to 0.86 ppm) during the period studied; hospital admissions were clearly elevated at a time when concentrations had not yet exceeded 715 μg/m^3 (0.25 ppm). Smoke shade was close to 3 coh units.

In London, a one-day exposure to a daily average level of 600 μg/m^3 of sulfur dioxide (0.20 ppm) caused accentuation of symptoms in persons with chronic respiratory disease on the day following the high sulfur dioxide level if particulate matter at a substantial concentration was also a pollutant.

This finding in London was also observed in a Chicago study. The Chicago study noted a sharp rise in illness rates on the day following a one-day exposure to 715 μg/m^3 (0.25 ppm) of sulfur dioxide or more for patients 55 years of age and over with severe bronchitis. Particulate matter was also present.

Effects at lower levels of sulfur dioxides have also been noted. In Rotterdam, during a few days in which sulfur dioxide concentrations rose from about 300 to 500 μg/m^3 (0.11 ppm to 0.19 ppm), the number of hospital admissions for irritations of the respiratory system rose, particularly for older persons. Absenteeism from work under such conditions increased substantially, especially for those 45 years old and over.

The lowest levels at which effects are reported for short-time periods are those reported for the Ruhr area. An effect was noted on "functional disturbance" at an estimated daily indoor mean of 270 μg/m^3 (0.10 ppm) for 4 days and an increase in "symptoms, illnesses, or diseases" (breathlessness, throat and eye irritation, and "depression and apathy without further specification") was noted at a daily indoor mean of about 540 μg/m^3 (0.20 ppm).

Longer term exposure to lower levels than those found during an air pollution episode has also been associated with demonstrable health effects. It is for this reason that it must be emphasized that the levels of a pollutant at which effects are detected are not the concentrations at which the pollutant may begin to have an effect on health. The initiation of the deleterious effects presumably must take place before, and at a lower concentration, than that at which the existence of a strong association is accepted for statistical reasons. Repeated respiratory infections in early childhood, for example, which in one study appeared to be related to air pollution, may have contributed to the later development of the chronic bronchitis syndrome in the adult.

A major British study has found an association between mortality from bronchitis and lung cancer and levels of air pollution, after taking into consideration differences in age, smoking habits, social class, and occupational exposure. The sulfur dioxide values for a year averaged 116 μg/m^3 (0.040 ppm) for the polluted area, and 75 μg/m^3 (0.026 ppm) for the cleaner area. Corresponding smoke values were 160 μg/m^3 and

80 $\mu g/m^3$, respectively. It was not possible to separate the effects of sulfur dioxide from the effects of comparable amounts of smoke which were present. This is consistent with other British studies, which indicate an association of bronchitis death rates with air pollution concentrations that is apparently independent of social class differences, but which can not specify the specific role played by sulfur oxides.

Children and housewives appear to represent the most suitable subjects for determining the health effects of long-term exposures to routine levels of air pollution. A study in Genoa, Italy, included housewives 65 or more years of age, who were non-smokers, and who had lived for a long period in the same area without having any industrial experience. Sulfur dioxide was monitored for 10 years at 19 sites by a technique analagous to the volumetric method of the British. An increased frequency of cough, sputum, dyspnea, and bronchitis was noted in the moderately polluted area as compared to the relatively clean area. Differences were noted in the summer prevalence of respiratory diseases in the industrial area, with an annual mean of 265 $\mu g/m^3$ SO_2 (0.093 ppm) when compared to the middle [annual mean of 105 $\mu g/m^3$ SO_2 (0.037 ppm)] and low [annual mean of 80 $\mu g/m^3$ SO_2 (0.028 ppm)] pollution areas. The study also showed a very significant correlation between the frequency of bronchitis and the annual mean of sulfur dioxide levels for the seven districts of the city, whereas the correlation between the frequency of bronchitis and suspended matter and dustfall was not significant.

Another morbidity study which has included housewives is that conducted in Nashville. This showed a direct correlation between illnesses for all causes for housekeeping white females, 15 to 64 years of age, and sulfur dioxide levels. It also showed that the cardiovascular morbidity in the 55 and older age group, for both sexes, was twice as high in the most polluted area as compared with the least. Cigarette smoking was not taken into account, and the adjustment made for socioeconomic status may not have been wholly adequate.

Differences in respiratory symptoms have also been found in areas which had long-term values of 95 $\mu g/m^3$ sulfur dioxide (0.034 ppm) and 25 $\mu g/m^3$ (0.009 ppm) measured by hydrogen peroxide absorption, sulfuric acid titrations. The study is unusual in that the source of pollution was the single stack of a smelter, and daily averages were up to 17 times the annual mean. However, the findings that respiratory symptoms disappeared when pollution was locally reduced through installation of a higher stack, would appear to confirm an effect of sulfur dioxide at the concentrations originally measured, provided that no other change took place which could account for the disappearance of symptoms.

Studies of schoolchildren in Great Britain have indicated that increased frequency and severity of respiratory diseases occurred when long-term pollution levels exceeded averages of about 120 $\mu g/m^3$ (0.046 ppm) sulfur dioxide and 100 $\mu g/m^3$ for smoke.

4. Effects on Visibility

Particles suspended in the air reduce visibility, or visual range, by scattering and absorbing light coming from both an object and its background, thereby reducing the contrast between them. Moreover, suspended particles scatter light into the line of sight, illuminating the air between, to further degrade the contrast between an object and its background. This phenomenon is described in detail in a companion document *Air Quality Criteria for Particulate Matter.*

The scattering of light into and out of the line of viewing by particles in the narrow range of 0.1 μ to 1 μ in radius has the greatest effect on visibility. Of the total suspended particulate matter in urban air, commonly from 5 percent to 20 percent consists of sulfuric acid and other sulfates, and of these, 80 percent or more by weight are smaller than 1 μ in radius. Consequently, suspended sulfates in the air can contribute significantly to reduction in visibility.

Characteristic behavior of suspended particles in the size range mentioned makes it possible to relate visual range to concentrations of overall particulate matter. Since sulfur dioxide levels, in general, correlate with levels of overall suspended particulate matter, and since the ratio of sulfur dioxide to suspended sulfate can be estimated, given the relative humidity, it is possible to estimate visibility for various relative humidities from sulfur dioxide concentration. Using such data as appear in Chapter 1, Figure 1–5, we can estimate that at a concentration of 285 $\mu g/m^3$ (0.10 ppm) of sulfur dioxide and with a relative humidity of 50 percent, visibility in New York City would typically be reduced to about 5 miles. At a visual range of less than 5 miles, operations are slowed at airports because of the need to maintain larger distances between aircraft. Federal Aviation Administration restrictions on aircraft operations become increasingly severe as the visual range decreases below 5 miles.

5. Effects on Materials

Laboratory and field studies underscore the importance of the combination of particulate and sulfur oxides pollution in a wide range of damage to materials. On the basis of present knowledge, it is difficult to evaluate precisely the relative contribution of each of the two classes of pollution; however, some general conclusions may be drawn.

Steel test panels, dusted with a number of active hygroscopic particles commonly found in polluted atmospheres, corroded at a low rate in clean air at relative humidities below 70 percent. The corrosion rate was higher at relative humidities above 70 percent. It greatly increased when traces of sulfur dioxide were added to the laboratory air.

It is apparent that corrosion rates of various metals are higher in urban and industrial atmospheres with relatively high levels of both particulate and sulfur oxides than they are in rural and other areas of low pollution. High humidity and temperature also play an important synergistic part in this corrosion reaction. Studies show increased corrosion rates in industrial areas where air pollution levels, including sulfur oxides and particulates, are higher. Further, corrosion rates are higher during the fall and winter seasons when particulate and sulfur oxides pollution is more severe, due to a greater consumption of fuel for heating. Depending on the kind of metal exposed as well as location and duration of exposure, corrosion rates were 1½ to 5 times greater in polluted atmospheres than in rural environments.

In Chicago and St. Louis, where steel panels were exposed at a number of sites, high correlations were found in each city between corrosion rates, as measured by weight loss, and sulfur dioxide concentrations, as measured by the West-Gaeke method. In St. Louis, except for one exceptionally polluted site, corrosion losses were 30 percent to 80 percent higher than losses measured in nonurban locations. Sulfation rates in St. Louis, measured by lead peroxide candle, also correlated well with weight loss due to corrosion. Measurements of dustfall in St. Louis, however, did not correlate significantly with corrosion rates. Over a 12-month period in Chicago, the corrosion rate at the most corrosive site (mean SO_2 level of 0.12 ppm) was about 50 percent higher than at the least corrosive site (mean SO_2 level of 0.03 ppm). Although suspended particulate levels measured in Chicago with high-volume samplers also correlated with corrosion rates, a co-variance analysis indicated that sulfur dioxide concentrations were the dominant influence on corrosion. Based on these data, it appears that considerable corrosion may take place (i.e., from 11 percent to 17 percent weight loss in steel panels) at annual average sulfur dioxide concentrations in the range of 0.03 ppm to 0.12 ppm, and although high particulate levels tend to accompany high sulfur dioxide levels, the sulfur dioxide concentration appears to have the more important influence.

Sulfur oxides pollution contributes to the damage of electrical equipment of all kinds. Studies have reported a one-third reduction in the life of overhead power line hardware and guy wires in heavily polluted areas. In some areas it has been found necessary to use more expensive, less corrodible metals, such as gold, for electrical contacts.

Sulfur oxides pollution attacks a wide variety of building materials—limestone, marble, roofing slate, and mortar—as well as statuary and other works of art, causing discoloration and deterioration. Certain textile fibers—such as cotton, rayon, and nylon—are harmed by atmospheric sulfur oxides.

Dyed fabrics may fade in atmospheres containing sulfur oxides and other pollutants. Severe fading was noted for some dyes in fabrics exposed in Chicago, where annual average sulfur dioxide levels were 0.09 ppm. Leather exposed to sulfur oxides may lose much of its strength, and paper may become discolored and brittle.

Concentrations of 1 ppm sulfur dioxide can increase the drying time of some oil-based paints by 50 percent to 100 percent. Some films become softer and others more brittle, both developments adversely affecting durability. Sulfur dioxide also appears to render some paint films water sensitive, consequently reducing the film gloss. Under certain conditions sulfur dioxide levels of 0.1 ppm to 0.2 ppm cause the blueing of Brunswick green, and in the presence of ammonia produce a troublesome defect called crystalline bloom brought about by the formation of very small ammonium sulfate crystals.

6. Effects on Vegetation

Sulfur dioxide may cause acute or chronic leaf injury to plants. Acute injury, produced by high concentrations for relatively short periods, usually results in injured tissue drying to an ivory color; it sometimes results in a darkening of the tissue to a reddish-brown. Chronic injury, which results from lower concentrations over a number of days or weeks, leads to pigmentation of leaf tissue, or leads to a gradual yellowing, or chlorosis, in which the chlorophyll-making mechanism is impeded. Both acute and chronic injury may be accompanied by the suppression of growth and yield.

Acute injury apparently affects the plant's ability to transform absorbed sulfur dioxide into sulfuric acid, and then into sulfates. At high rates of absorption, sulfite is thought to accumulate, resulting in the formation of sulfurous acid, which attacks the cells. The amount of acute injury depends on the absorption rate, which is a function of the concentration. A given amount of gas at a high concentration will be absorbed in a shorter period and will cause more leaf destruction than the same amount of gas at a lower concentration. Mathematical expressions have been worked out which, for some plant species, relate concentration, time of exposure, and amount of damage.

Different varieties of plants vary widely in their susceptibility to acute sulfur dioxide injury. The threshold response of alfalfa to acute injury is 1.25 ppm over 1 hour, whereas privet requires 15 times this concentration for the same amount of injury to develop. Some species of trees and shrubs have shown injury at exposures of 0.5 ppm for 7 hours, while injury has been produced in other species at 3-hour sulfur dioxide exposures of 0.54 ppm and, in still others, at 8-hour exposures of 0.3 ppm. From such studies, it appears that acute symptoms will not occur if the 8-hour average concentration does not exceed 0.3 ppm. (From the data on the CAMP cities, a maximum 8-hour concentration of 0.3 ppm would correspond to a yearly average concentration of between 0.03 ppm and 0.05 ppm). However, sulfur dioxide concentrations from 0.05 to 0.25 ppm may react synergistically with either ozone or nitrogen dioxide in short-term exposures (e.g., 4 hours) to produce moderate-to-severe injury to certain sensitive plants.

Chronic plant injury results from the gradual accumulation of excessive amounts of sulfate in leaf tissue. Sulfate formed in the leaf is additive to sulfate absorbed through the roots, and when sufficiently high levels accumulate, chronic symptoms, accompanied by leaf drop, may occur. Chronic symptoms and excessive leaf drop have been reported in locations where the mean annual concentration is below approximately 0.03 ppm.

It has been suggested that sulfur dioxide might suppress growth and yield without causing visible injury. One investigator reported that yields of rye grass grown in unfiltered air were significantly lower than similar yields of plants grown in filtered air. No visible symptoms of injury were observed. Sulfur dioxide levels in the unfiltered air ranged from 0.01 ppm to 0.06 ppm, with exposure periods ranging from 46 to 81 days; other gaseous pollutants may have also been present. Usually, the suppression of growth and yield is accompanied by visible symptoms of injury—a linear relationship has been derived, for example, between the yield of alfalfa and the total area destroyed by acute symptoms, or the area covered by chlorosis.

Sulfuric acid mist, which may occur in polluted fogs and mists, also damages leaves. The acid droplets may cause a spotted injury to wet leaves at concentrations of 0.1 mg/m^3.

B. CONCLUSION

The conclusions which follow are derived from a careful evaluation by the National Air Pollution Control Administration of the foreign and American studies cited in previous chapters of this document. They represent the Administration's best judgment of the effects that may occur when various levels of pollution are reached in the atmosphere. The data from which the conclusions were derived, and qualifications which should be considered in using the data, are identified by chapter reference in each case.

1. Effects on Health

Analyses of numerous epidemiological studies clearly indicate an association between air pollution, as measured by sulfur dioxide, accompanied by particulate matter, and health effects of varying severity. This association is most firm for the short-term air pollution episodes.

There are probably no communities which do not contain individuals with impaired health who are particularly susceptible to the adverse effects of elevated levels of sulfur oxides and particulate matter. However, to show small changes in deaths associated with coincident higher levels of air pollutants requires extremely large populations. In small cities, these changes are difficult to detect statistically.

The epidemiologic studies concerned with increased mortality also show increased morbidity. Again, increases in morbidity as measured, for example, by increases in hospital admissions or emergency clinic visits, are most easily detected in major urban areas.

It is believed that, for the large urban communities which are routinely exposed to relatively high levels of pollution, sound statistical analysis can detect with confidence the small changes in daily mortality which are associated with fluctuation in pollution concentrations. Such analysis has thus far been attempted only in London and in New York.

The association between long-term community exposures to air pollution and respiratory disease incidence and prevalence rates is conservatively believed to be intermediate in its reliability. Because of the reenforcing nature of the studies conducted to date, the conclusions to be drawn from this type of study can be characterized as probable.

The association between long-term residence in a polluted area and chronic disease morbidity and mortality is somewhat more conjectural. However, in the absence of other explanations, the findings of increased morbidity and of increased death rates for selected causes, independent of economic status must still be considered consequential.

Based on the above guidelines the following conclusions are listed in order of reliability, with the more reliable conclusions first.

As discussed in Chapter 2, the sulfur oxides measurement systems used by American and foreign agencies are not always the same. However, for the most part, data derived from one measurement system can be converted to other systems.

a. AT CONCENTRATIONS OF ABOUT *1500 μg/m^3* (0.52 ppm) of sulfur dioxide (24-hour average), and suspended particulate matter measured as a soiling index of 6 cohs or greater, *increased mortality* may occur. (American data; see Chapter 9, Section C–1a.)

b. AT CONCENTRATIONS OF ABOUT *715 μg/m^3* (0.25 ppm) of sulfur dioxide and higher (24-hour mean), accompanied by smoke at a concentration of 750 μg/m^3, *increased daily death rate* may occur. (British data; see Chapter 9, Section C–1a.)

c. AT CONCENTRATIONS OF ABOUT *500 μg/m^3* (0.19 ppm) of sulfur dioxide (24-hour mean), with low particulate levels, *increased mortality rates* may occur. (Dutch data; see Chapter 9, Section C–1a.)

d. AT CONCENTRATIONS RANGING FROM *300 μg/m^3 to 500 μg/m^3* (0.11 ppm to 0.19 ppm) of sulfur dioxide (24-hour mean), with low particulate levels, *increased hospital admissions* of older persons for respiratory disease may occur; *absenteeism* from work, particularly with older persons, may also occur. (Dutch data; see Chapter 9, Section C-1b.)

e. AT CONCENTRATIONS OF ABOUT *715 $\mu g/m^3$* (0.25 ppm) of sulfur dioxide (24-hour mean), accompanied by particulate matter, *a sharp rise in illness rates* for patients over age 54 with severe bronchitis may occur. (American data; see Chapter 9, Section C-5.)

f. AT CONCENTRATIONS OF ABOUT *600 $\mu g/m^3$* (about 0.21 ppm) of sulfur dioxide (24-hour mean), with smoke concentrations of about 300 $\mu g/m^3$, patients with chronic lung disease may experience *accentuation of symptoms*. (British data; see Chapter 9, Section C-5.)

g. AT CONCENTRATIONS RANGING FROM *105 $\mu g/m^3$ to 265 $\mu g/m^3$* (0.037 ppm to 0.092 ppm) of sulfur dioxide (annual mean), accompanied by smoke concentrations of about 185 $\mu g/m^3$, *increased frequency of respiratory symptoms and lung disease* may occur. (Italian data; see Chapter 9, Section C-2.)

h. AT CONCENTRATIONS OF ABOUT *120 $\mu g/m^3$* (0.046 ppm) of sulfur dioxide (annual mean), accompanied by smoke concentrations of about 100 $\mu g/m^3$, *increased frequency and severity of respiratory diseases* in schoolchildren may occur. (British data; see Chapter 9, Section C-3.)

i. AT CONCENTRATIONS OF ABOUT *115 $\mu g/m^3$* (0.040 ppm) of sulfur dioxide (annual mean), accompanied by smoke concentrations of about 160 $\mu g/m^3$, *increase in mortality* from bronchitis and from lung cancer may occur. (British data; see Chapter 9, Section C-2.)

2. Effects on Visibility

AT A CONCENTRATION OF *285 $\mu g/m^3$* (0.10 ppm) of sulfur dioxide, with comparable concentration of particulate matter and relative humidity of 50 percent, *visibility may be reduced* to about five miles. (American data; see Chapter 1, Figure 1-3.)

3. Effects on Materials

AT A MEAN SULFUR DIOXIDE LEVEL OF *345 $\mu g/m^3$* (0.12 ppm), accompanied by high particulate levels, the *corrosion rate* for steel panels may be increased by 50 percent. (American data; see Chapter 4, Section C.)

4. Effects on Vegetation

a. AT A CONCENTRATION OF ABOUT *85 $\mu g/m^3$* (0.03 ppm) of sulfur dioxide (annual mean), *chronic plant injury* and *excessive leaf drop may occur.* (Canadian data; see Chapter 5, Section C.)

b. AFTER EXPOSURE TO ABOUT *860 $\mu g/m^3$* (0.3 ppm) of sulfur dioxide for 8 hours, some species of trees and shrubs show *injury*. (American data; see Chapter 5, Section C.)

c. AT CONCENTRATIONS OF ABOUT *145 $\mu g/m^3$* to *715 $\mu g/m^3$* (0.05 ppm to 0.25 ppm), sulfur dioxide may react synergistically with either ozone or nitrogen dioxide in short-term exposures (e.g., 4 hours) to produce *moderate to severe injury* to sensitive plants. (American data; see Chapter 5, Section E.)

C. RESUME

In addition to health considerations, the economic and aesthetic benefits to be obtained from low ambient concentrations of sulfur oxides as related to visibility, soiling, corrosion, and other effects should be considered by organizations responsible for promulgating ambient air quality standards. Under the conditions prevailing in areas where the studies were conducted, adverse health effects were noted when 24-hour average levels of sulfur dioxide exceeded 300 $\mu g/m^3$ (0.11 ppm) for 3 to 4 days. Adverse health effects were also noted when the annual mean level of sulfur dioxide exceeded 115 $\mu g/m^3$ (0.04 ppm). Visibility reduction to about 5 miles was observed at 285 $\mu g/m^3$ (0.10 ppm); adverse effects on materials were observed at an annual mean of 345 $\mu g/^3$ (0.12 ppm); and adverse effects on vegetation were observed at an annual mean of 85 $\mu g/m^3$ (0.03 ppm). It is reasonable and prudent to conclude that, when promulgating ambient air quality standards, consideration should be given to requirements for margins of safety which take into account long-term effects on health, vegetation, and materials occurring below the above levels.

APPENDICES

APPENDIX A—SYMBOLS

A	the cross sectional area of a particle for light attenuation
A_{ij}	the cross sectional area of an i particle (see i and j, below)
C	the sulfur dioxide concentration (ppm or $\mu g/m^3$) in air or the weight (percent) H_2SO_4 in acid droplets
C_o	the minimum concentration which will damage a particular plant in a given time
E	the particle-scattering ratio
E_{ij}	the scattering ratio of an i particle
K	a constant
L_v	visual range
M_g	geometric mean. For sample values x_1, x_2, . . . x_n, the geometric mean is $\sqrt[n]{x_1, x_2, \ldots x_n}$ or $\left\{ \prod_{j=1}^{n} x_j \right\}^{\frac{1}{n}}$
N	the number of particles per unit volume of atmosphere
N_{ij}	the number of ij particles per unit volume
P	probability
S_{35}	the radioactive isotope of sulfur having a mass number 35
a	a constant related to damage to a particular plant species by sulfur dioxide
d	the diameter of a spherical particle
e	Napierian log base (=2.718281)
f	the fraction of pollutant gases replaced per unit time with diluting air
g	the acceleration of gravity, cm/sec^2
i	(subscript) identifies a particle of a given diameter and a given index of refraction
j	identifies a particle of a given refractive index
k	the fraction of SO_2 loss per unit time due to oxidation to SO_3
n	the index of refraction
p	the coefficient for converting L_v to desired units
r	a constant related to the minimum SO_2 concentration that damages a particular species of plant
r	correlation coefficient
t	the exposure time
z	"standard normal exceedance deviate". The number of standard deviations an observed value is from the median when the underlying distribution is normal.
λ	the wavelength of light in Ångstroms, microns, or nanometers
ρ	the density of a particle or droplet
σ	the attenuation coefficient per unit path length
σ_g	standard geometric deviation
x	probability function, usually expressed as x^2 ("CHI SQUARE")
Σ	summation, or sum of a series

APPENDIX B—ABBREVIATIONS

Å	Ångstrom, $1\ \text{Å} = 10^{-8}$ cm
CAMP	Continuous Air Monitoring Project
coh	coefficient of haze
cm	centimeter, $1\ \text{cm} = 10^{-2}$ m
CMD	count median diameter
cm^2	square centimeter
DSIR	Department of Scientific and Industrial Research (England)
FEV	forced expiratory volume
FVC	forced vital capacity
g	mass, grams
hr	hour
LC_{50}	concentration of toxicant lethal to 50% of subjects
l	volume, liters
MMD	mass median diameter
m	length, meters
m^3	cubic meter
mi	mile
mi^2	square mile
min	minutes
μ	micron, $1\mu = 10^{-4}\ \text{cm} = 10^{-6}$ m
μg	microgram, $1\ \mu\text{g} = 10^{-6}$ g
mg	milligram, $1\ \text{mg} = 10^{-3}$ g
MRC	Medical Research Council
mμ	millimicron, $1\ \text{m}\mu = 10^{-9}\ \text{m} = 1\text{nm}$
NASN	National Air Surveillance Network
nm	nanometer, $1\text{nm} = 10^{-9}\ \text{m} = 1\text{m}\mu$
mo	months
ppb	parts per billion
ppm	parts per million
pphm	parts per hundred million
RH	relative humidity
sec	seconds
yr	year

APPENDIX C—CONVERSION FACTORS

To Convert	*To*	*Multiply By*
mg/m^3	μg/m^3	1000
mg/100m^3	μg/m^3	10
μg SO_2/m^3 (0° C, 760mm Hg)	ppm SO_2 (vol)	3.5×10^{-4}
μg SO_2/m^3 (0° C, 760mm Hg)	ppm SO_2 (wgt)	7.7×10^{-4}
ppm SO_2 (vol)	μg SO_2/m^3 (0° C, 760 mm Hg)	2860
ppm SO_2 (wgt)	μg SO_2/m^3 (0° C, 760 mm Hg)	1290
μ or μm	cm	10^{-8}
μ	cm	10^{-4}
Å	ft	3.28
μ ??	g	453.6
m ??	m	~~10^{-6}~~ = 1,000
lb	Å	10^{-4}

APPENDIX D—GLOSSARY

Abscission—the natural separation of flowers, fruit, and leaves from plants by the development and subsequent disorganization of a separation layer

Aerosol—a cloud of solid particles and/or liquid droplets smaller than 100μ in diameter, suspended in a gas

Airway—any part of the respiratory tract through which air passes during breathing

Airway resistance—resistance to the flow of air in the passages to the lungs

Ala (pl. *alae*) *nasi*—the outer part of the nostril

Alpha rhythm—a uniform set of electroencephalographic waves with a frequency of approximately ten per second

Alveolus (pl. alveoli)—a small, sac-like dilation at the inner most end of the airway, through whose walls gaseous exchange takes place

Anemia—a condition in which the number of red blood cells per cubic centimeter of blood is below normal; can be further characterized by descriptions of the alterations in the red blood cells (e.g., *hypochromic, microcytic*) or of the clinical state it is associated with

Anhydride—a chemical compound derived by the extraction of a molecule of water from the original molecule

Anoxia—a deficiency of the amount of oxygen reaching the body tissues

Arrest, cardiac—the cessation of heartbeat

Atelectasis—the collapse of all or part of a lung, with resultant loss of functioning tissue

Atropine—a parasympatholytic drug which in general tends to relax smooth muscle, slow the heart rate, and dilate the pupils

Attenuation—in physics, any process in which the flux density (or power, amplitude, intensity, illuminance, etc.) of a "parallel beam" of energy decreases with increasing distance from the energy source. A more general usage of this word is also found in this publication: any reduction in strength, density, effect, or amplitude.

Auscultation (adj. *auscultatory*)—the act of listening for sounds within the body, usually with the use of a stethoscope

Bloom, crystalline—the formation of very small crystals on the surface of organic films, resulting in noticeable bloom caused by the efficient scattering of light

Bradycardia—an abnormal slowness of the heartbeat

Bronchiectasis—a chronic dilatation of a bronchial passage

Bronchiole—one of the finer subdivisions of the bronchial tree

Bronchitis—an inflammation of the bronchi, usually manifested clinically by cough and the production of sputum

Bronchitis, chronic—a long-standing inflammation of the bronchi characterized by excessive mucus secretion in the bronchial tree and manifested by a persistent or recurrent productive cough. For the purposes of definition, these symptoms must be present on *most days* for a minimum of *3 months* of the year for at least *2* successive *years* (American Thoracic Society)

Bronchoconstriction—a diminution in the size of the lumen of a bronchus

Bronchodilatation—an increase in the size of the lumen of a bronchus

Bronchus (pl. *bronchi*)—one of the larger air passages in the lung

Buffer—a substance capable of enabling a system to resist changes in condition; a solution whose pH is changed only slightly by the addition, within limits, of an acid or a base

Cannula—a small tube for insertion into a body cavity or into a duct or vessel

Capacity, forced vital (FVC)—the largest amount of gas which can be forcibly expired from the lungs following a maximal inspiration

Capacity, functional residual (FRC)—the volume of gas remaining in the lungs as the resting end-expiratory level

Capacity, vital—the maximum volume of gas which can be expired from the lungs following a maximum inspiration

Carcinogenesis—the production of cancer

Carina—a ridgelike structure; in anatomy of the respiratory tract, it is a prominence on the lowest tracheal cartilage and is situated between the orifices of the two mainstem bronchi

Catalyst (adj. *catalytic*)—a substance capable of increasing the velocity of a reaction without chemically or physically changing itself

Catheter—a tubular device inserted into a canal, vessel, or body cavity for either the introduction or extraction of some material

Chloroplast—a specialized body (a plastid) containing chlorophyll in the cytoplasm of plants; the site of photosynthesis and starch formation in plants

Chlorosis—a disease condition in green plants, marked by yellowing or blanching

Chronaxie (chronaxy)—the minimum time required for the excitation of a nervous element by a specified stimulus

Cilium (pl. *cilia*)—small, hairlike process attached to a free surface of a cell, capable of rhythmic movement

Clearance—the removal of material from the body or from an organ

Concentration—the total mass (usually in micrograms) of the suspended particles contained in a unit volume (usually one cubic meter) at a given temperature and pressure; sometimes, the concentration may be expressed in terms of total *number* of particles in a unit volume (e.g., parts per million); concentration may also be called the "loading" or the "level" of a substance; concentration may also pertain to the strength of a solution

Conductometric—a method of analysis based on the conductivity of a sample

Conifer—belonging to the coniferales order, consisting primarily of evergreen trees and shrubs

Conjunctiva—the delicate membrane lining the eyelids and covering the exposed surface of the eyeball

Conjunctivitis—an inflammation of the conjunctiva

Consolidation—the process by which a diseased lung passes from an aerated collapsible state to one of an airless solid consistency because of accumulation of exudate

Criteria, air quality—a compilation of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects

Deciduous—falling off or shed at the end of a growing period or season

Deliquesce—to dissolve gradually and become liquid by absorbing moisture from the air

Desorption—the release of a substance which has been taken into another substance by a physical process or held in concentrated form upon the surface of another substance; the reverse of absorption or adsorption

Desquamate—to cast off epidermis in shreds or scales; to peel off in sheets or scales

Diameter, count median (CMD)—the geometric median size of a distribution of particles, based on a numerical count

Diameter, mass median (MMD)—the geometric median size of a distribution of particles, based upon a weight (usually derived from a Stokes' Diameter)

Dichotomous—dividing in succession into pairs; showing a dual arrangement

Distal—furthest or most remote from the median line of the body, from the point of attachment, or from the origin; peripheral (*cf.* proximal)

Dolomite—a limestone or marble rich in magnesium carbonate

Dyspnea—difficult or labored breathing

Edema—a condition due to the presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body

Effusion—the escape of fluid into a tissue or part, usually by the rupture of a vessel or extravasation through its walls

Electroencephalogram (EEG)—a graphic recording of electric currents developed in the brain, obtained with the use of electrodes which are usually applied to the scalp

Emphysema—a swelling due to the presence of air, usually excess or additional air. The term is usually used to refer to pulmonary emphysema

Emphysema, pulmonary—a condition in which there is overdistension of air spaces and resultant destruction of alveoli and loss of functioning lung tissue

Epidemiology—a science dealing with the factors involved in the distribution and frequency of a disease process in a population

Epidermis—the outermost layer of skin in animals; any integument

Epithelium—a closely packed sheet of cells arranged in one or more layers, covering the surface of the body and lining hollow organs

Exacerbation—an increase in the severity of any symptoms or of a disease

Exteroceptive—activated by or relating to any stimulus impinging on an organism from the outside

Fibrosis—the development of fibrous tissue; sclerosis

Filiform—having the shape of a thread or filament

Follicle (follicule)—a very small excretory or secretory sac or a small gland

Gallamine—a drug used to relax skeletal muscles

Gneiss—a laminated or foliated metamorphic rock

Goblet cell—a type of epithelial cell containing mucus and having the shape of a flask or goblet

Halide—any binary compound of a halogen

Halogen—any one of the chemically related elements fluorine, chlorine, bromine, iodine, and astatine

Hemoglobin—a protein found in red blood cells, responsible for oxygen transport to all parts of the body

Hilum (hilus)—a depression or pit at that part of an organ where the vessels and nerves enter

Histamine—a substance which produces dilatation of capillaries and stimulates gastric secretion, occurring in both animal and vegetable tissues; β-imidazol-ethylamine

Histology—the study of the anatomy of tissues and their microscopic cellular structure

Hydrocarbon—a compound containing only hydrogen and carbon. This group is subdivided into alicyclic, aliphatic, and aromatic hydrocarbons according to the arrangement of the atoms and the chemical properties of the compounds

Hygroscopic—readily absorbing and retaining moisture

Hypertrophy—an enlargement or overgrowth of an organ or tissue due to an increase in the size of its constituent cells

Impactor, cascade—an instrument which employs several impactions in series to collect successively smaller sizes of particles

Incidence—the rate at which a certain event or disease occurs

Intercostal—situated between ribs

Interoceptive—of or relating to any stimulus arising from within the body

Interstitial—pertaining to or situated in the space between cells

Isopleth—a line on a map connecting points at which a given variable has a specified constant value

Isoproterenol—a sympathomimetic drug which is used to relieve bronchoconstriction and which also can function as a cardiac stimulant

Lacrimation (lachrymation)—tear formation, especially in excess

Lamina propria—a connective tissue layer located just beneath the epithelial cells and basement membrane of many organs

Larch—a tree of the genus *Larix* or of the family Pinaceae

Larynx—the organ concerned with the production of the voice, situated at the upper end of the trachea

Leach—to dissolve out by the action of a percolating liquid

Lesion—an injury or other circumscribed pathologic change in a tissue

Lumen—the inner space of a hollow organ or tube

Lymphocyte—a variety of white blood cells which arises in the reticular tissue of lymph glands

Lymphoid cell—a mononuclear cell found in lymphoid tissue (such as the spleen, tonsil, lymph nodes), ultimately concerned with immunologic function

Mean, geometric (Mg)—a measure of central tendency for a log-normal distribution; the value of a given set of samples above which 50 percent of the values lie

Monochromatic light—a beam of light having some desired, narrow range of wavelengths

Monodisperse—characterized by particles of uniform size in a dispersed phase

Morbidity—the occurrence of a disease state

Morphology—a branch of biology dealing with the structure and form of living organisms

Mortaliy—the ratio of the total number of deaths to the total population, *or* the ratio of the number of deaths from a given disease to the total number of people having that disease

Mucosa—a mucous membrane

Mucus (adj. *mucous*)—the clear viscid secretion of a mucous membrane

Mural—pertaining to the wall of a cavity

Nasopharynx—the part of the pharynx (throat) lying above the level of the soft palate

Nebulize—to reduce to a fine spray

Necrosis—localized death of cells

Nembutal—a barbiturate drug used as a hypnotic and a sedative; pentobarbital

Node—a circumscribed swelling

Node, lymph—one of many accumulations of lymphatic tissue situated throughout the body

Nucleus (condensation nucleus)—a particle in the size range from 0.1μ to 1μ which serves as a nidus on which water or other vapors in the air can condense to form liquid droplets

Olefin—a class of unsaturated hydrocarbons of the general formula $C_n H_{2n}$

Olfactory—pertaining to the sense of smell

Optical—pertaining to vision

Oropharynx—that part of the pharynx (throat) lying between the level of the soft palate and the epiglottis

Paraffin—a purified mixture of solid hydrocarbons obtained from petroleum, occurring as an odorless, tasteless, colorless or white, relatively translucent mass

Parenchyma—the specific or functional tissue of a gland or organ, as opposed to its supporting framework

Pathogen—any disease-producing organism or material

Pathogenesis—the production or the mode of origin and development of a disease condition

Pathology—the study of the essential nature of disease, particularly with respect to the structural and functional changes in organs and tissues

Pharynx—the upper expanded portion of the alimentary canal lying between the mouth, the nasal cavities, and the beginning of the esophagus; the throat

Photochemistry—a branch of chemistry dealing with the effect of radiant energy (as light) in producing chemical changes

Photosynthesis—the formation of carbohydrate from carbon dioxide and water in the presence of chlorophyll and light, in plant tissues

Physiology—a science which studies the function of a living organism or its parts

Phytotoxic—harmful to plant materials

Plethysmograph—an apparatus for the determination and recording of a change in the size of an organ or limb or body

Pneumonitis—a general term for inflammation of the lung

Pneumotachygraph—an instrument used to determine the force and velocity of respired air

Potentiation—synergism, as between two agents which together have a greater effect than the sum of their effects when acting separately

Prevalence—the number of cases of a disease at a given time

Privet—any one of various plants of the genus *Ligustrum*, used extentively as ornamental shrubs

Procaine—a drug which is used primarily as a local anesthetic

Proximal—nearest to the center of the body or the point of origin (*cf.* distal)

Rale—an abnormal respiratory sound heard in auscultation of the chest

Ratio, standardized mortality—the ratio of the number of deaths observed in a given population over a given period of time *to* the number of deaths expected to occur in the given population over the same period of time if the given population behaved as any other group of similar composition would during that same period

Rhinitis—an inflammation of the nasal mucous membrane

Rhinorrhea—"runny nose"

Rhonchus (*pl.* rhonchi)—a dry, coarse sound usually originating from partial obstruction in a bronchial tube

Spasm—an involuntary and abnormal muscular contraction, usually sudden and forceful, and often accompanied by pain and/or loss of function

Spectroscopy—the branch of physical science dealing with the theory and interpretation of bands of light

Spirometer—an instrument for the measurement of the volume of gas respired by the lungs

Squamous—resembling or covered with scales

Standards, air quality—levels of air pollutants which can not legally be exceeded during a specific time in a specific geographical area

Subcutaneous—beneath the skin

Sympathetic—referring to the *sympathetic trunk* or the entire *sympathetic nervous system*, concerned with the involuntary regulation of cardiac muscle, smooth muscles, and glands

Synergism—a situation in which the combined action of two or more agents acting together is greater than the sum of the action of these agents separately

Systemic—relating to the body as a whole, rather than to its individual parts

Toxicology—the study of poisons, including their preparation, identification, physiologic action, and antidotes

Trachea—windpipe; the airway extending from the larynx to the origin of the two mainstem bronchi

Tracheotomy—a surgical opening through the skin and muscles of the neck into the trachea

Transpiration—the emission of water vapor from the surface of plant leaves

Vagosympathetic—referring to the parts of the nervous system with innervation by the vagus nerve and innervation through the sympathetic nervous system

Vagus—pertaining to either of the pair of tenth (X) cranial nerves, which supply parasympathetic, visceral afferent, motor, and general sensory innervation

Valence—an integer representing the number of hydrogen or chlorine atoms which one atom of an element is capable of combining with

Ventilation, minute—the total volume of gas respired in one minute, i.e., the tidal volume multiplied by breaths per minute.

Viscus (pl. viscera)—any internal organ within a body cavity

Visual range—the distance, under daylight conditions, at which the apparent contrast between the specified type of target and its background becomes just equal to the threshold contrast of an observer, i.e., the distance at which it is just possible to see a dark object against the sky near the horizon

Volume, forced expiratory (FEV)—the volume of gas forcibly exhaled over a given time interval (usually measured in seconds) after maximum inspiration, e.g., $FEV_{1.0}$ for this measurement over a one second period

Volume, minute—same as minute ventilation

Volume, tidal—the volume of gas inspired or expired during each respiratory cycle

AUTHOR INDEX

Adams, D. F., 61, 66
Agnese, G., 129
Ahlquist, N. C., 13
Ajax, R. J., 55
Alekseeva, M. V., 23
Altman, P. L., 61
Alshuller, A. P., 8
Amdur, M. O., 5, 75, 76, 77, 78, 79, 80, 81, 83, 91, 92, 93, 94, 107, 108, 109, 110
Andelman S. L., 91
Anderson, A., 143, 144
Anderson, D. M., 25, 52, 53
Anderson, D. O., 91, 120, 131, 134, 137
Anderson, R. J. 91
Angel, J. H., 140
Ascher, L., 91
Aviado, D. M., 78
Axt, C. J., 9

Baetjer, A. M., 107
Ball, C. O. T., 82
Barber, F. R., 42
Baines, F., 53, 54
Balchum, O. J., 78, 82
Barkley, J. F., 51, 53, 55
Barnes, J. M., 91
Barrett, C. F., 19
Barringer, A. R., 22
Bates, D. V., 134
Battista, S. P., 77
Becker, W. H., 137
Bell, A., 91, 132, 140
Bellin, A., 24, 25 43
Belton, J., 122, 123
Benner, R. C., 53
Benson, F. B., 23
Benson, H. E., 5
Berry, C. R., 65
Bertramson, B. R., 45
Bhardwar, D. V., 51
Bienstock, D., 5
Bleasdale, J. K. A., 62, 65
Bokhoven, C., 22
Boone, R. E., 25
Booras, S. G., 24
Bracewell, J. M., 7
Bradley, W., 120, 122
Brady, N. O., 45
Brandt, C. S., 61, 62, 63
Brasser, L. J., 33, 40, 120, 122, 123, 125, 127, 129, 133, 136, 137, 139
Brasted, R. C., 5
Braverman, M. M., 125
Brennan, L., 61
Brewer, R. F., 61, 66
Brice, R. M., 25
Brooks, A. G. F., 11
Brown, D. A., 127
Brunn, L. W., 5
Buck, S. F., 127, 128
Buckman, H. O., 45
Buffalini, J. J., 8
Burdick, L. R., 51, 53, 55
Burgess, F. 75, 76, 106
Burgess S. G., 120, 128
Burgess, W. A., 143, 144
Burn, J. L., 129, 144, 136
Burton, G. G., 94, 110
Bushtueva K. A., 13, 23, 43, 76, 96, 97, 98, 99, 107
Bye, W. E., 19, 23, 42
Bystrova, T. A., 82

Calvert, J. G., x, 8
Cappel, J., 75
Carey, G. C. R., 139
Carnow, B. W., 140
Carpenter, S. B., 8, 9
Cartwright, J., 11
Cassell, E. G., 119, 125, 126
Catcott, E. J., 91
Catteral, M., 91
Chaney, A. L., 25, 43, 45
Charlson, R. J., 13
Cherkasov, YE. F., 96
Clancy, F. K., 22
Coffin, D. L., 91
Collier, C., 78
Comichi, S., 23
Commins, B. T., 9, 11, 24, 38, 43
Comroe, J. H., 94
Conlee, C. J., 55
Cooper, P., 74
Cooper, W. C, 91
Corn, M., 77, 80, 81, 94, 107, 110
Coste, J. H., 13, 42
Coughanowr, D. R., 7
Could, R. A., 61, 63
Courtier, G. B., 13, 42
Couy, C. J., 53
Cralley, L. V., 81, 96
Critchlow, J., 76
Crowley, D. 122, 123
Cuffe, S. T., 19
Cullumbine, H., 75, 76, 106

Daines, R. H., 61, 63
Dainton, F. S., 8
Dalhamn, T., 75, 81, 83
Daly, C., 128
Darley, E. F., 61, 66
Davis, T. R. A., 77
De Groot, I., 126
De Treville, R. T., 106
Devitofrancesco, G., 7
Dev Jain, K., 8
Diamond, J. R., 119, 125, 126
Dickerson, R. C., 23
Dittner, D. S., 61
Dohan, F. C., 135
Dorries, W., 63
Douglas, J. W. B., 137
Doyle, G. J., 7, 8
Drinker, P., 75, 76, 91, 94
Drolette, B. M., 125
Dubrovskaya, F. I., 96, 97
Dutra, F. R., 75
Dybicki, J., 78, 82

Elfers, L. A., 22
Elfimova, E. V., 142
Elliott, A., 138
Elliott, G. O., 82
Endow, N., 8
Engdahl, R. B., 19
Erhardt, C. L., 125

Fairbairn, A. S., 128
Farber, S. M., 91
Farmer, J. R., 23
Ferris, B. G., Jr., 134, 143, 144
Fesch, J., 81
Field, F., 123, 125
Firket, J., 119
Fish, B. R., 7
Fletcher, C. M., 140
Foran, M. R., 52
Forker, G. M., 10
Frank, N. R., 78, 82, 83, 92, 93, 95, 109, 110
Franklin, E. C., 61, 63

Frey, S. A., 21
Fried, M., 45

Gall, D., 7
Gartrell, F. E., 8, 9
Gee, J. B. L., 94, 110
Gerhard, E. R., 7
Gerstle, R. W., 19
Gibbons, E. V., 52
Gilbert, E. E., 7
Gilbert, P. T., 53
Glasser, M., 123, 125
Goetz, A., 8
Goldberg, C., 128
Goldring, I. P., 74
Golden, C. C., 23
Goldsmith, J. R., 82, 91, 133
Gore, A. T., 120, 128
Gorham E., 23, 45, 128
Graesser, F. E., 74
Graf, P., 81
Greenburg, L., 23, 74, 122, 125
Greenblatt J. H., 51
Greenwald, I., 91
Grollman, A., 98
Gross, P., 106
Gruber, C. W., 19
Gunn, F. D., 76

Hagstrom, R. M., 130
Hamming, W. J., 24, 25, 43
Handyside, A. J., 138, 139
Hangebrauck, R. P., 19
Hartzell, A., 73, 74
Haselhoff, E., 63
Heck, W. W., 61, 62, 63, 66
Hedgcock, G. G., 61 64
Heggestad, H. E., 66
Heimann, H., 91
Hemeon, W. C. L., 80
Hendricks, R. H., 61, 62, 63, 66, 76
Hendrickson, E. R., 19
Hepting, G. H., 65
Heyssel, R. M., 82
High, M. D., 19
Hill, A. B., 117
Hill, G. R., 61, 62, 63, 64, 66
Hill, J. D., 140
Hindawi, I. J., 66
Hochheiser, S., 23
Hodgman, C. D., 10
Holbrow, G. L., 51
Holland, W. W., 133, 138, 139
Holmes, J. A., 61, 62, 63
Horada, M., 62, 65
Horton, R. J. M., 130
Horvath, H., 13
Huey, N. A., 24
Huschke, R. E., 10

Ishikawa, K., 78
Ivin, K. J., 8

Jacobs, M. B., 5, 23, 125
Johnson, A. B., 62
Johnston, H. S., 8
Johnstone, G. F., 7
Johnstone, H. F., 7
Jones, J. L., 8
Joosting, P. E., 33, 40, 120, 122; 123, 125, 127, 129, 133, 136, 137, 142,
Junge C. E., 7, 45

Kanitz, S., 129
Kapalin, V., 142
Katz, M., 8, 19, 23, 42, 45, 61, 64, 65
Kopezynski, S. L., 8
Kramer, G. D., 23
Kuczynski, E. R., 22
Kurland, L. T., 126
Kehoe, R. A., 143, 144
Kenline, Pa. A., 25
Kensler, C. J. 77
Kent, D. C., 79
Kerr, H. P., 139
Kitzmiller, K., 143, 144
Knowelden, J., 138, 140

Liadlaw, S. A., 91
Landeau, E., 91, 130
Lange, N. A., 10
Larsen, R. I., 33, 38, 40, 46
Lawther, P. J., 91, 95, 122, 140
Leblanc, T. J., 143, 144
Lee, R. E., Jr., 9
Leighton, P. A., 8, 10
Leonard, A. G., 122, 123
Leong, K. J., 74, 75
Lepper, M. H., 140
Liberti, A., 7
Lieben, J., 25, 52, 53
Lindau, G., 63
Linzon, S. N., 42, 65
Lobova, E. K., 84
Law, M. J. D., 23
Lucas, D. H., 19
Ludman, W. F., 23
Ludwig, F. L., 9
Ludwig, J. H., 19, 20
Lunn, J. E., 138, 140
Lutsep, H., 7
Lyon, T. L., 45
Lyons C. J., 91
Lyubimov, N. A., 23

MacFarland, H. A., 74, 75
Machle, W. F., 143, 144
Mader, P. P., 24, 25, 43
Maeller T., 5
Manzhenko, E. G., 138
Markush, R. E., 127
Martin, A., 42
Martin, A. E., 91, 120, 122, 126

Mason, B. J., 7
Massey, L. M., 61
Mathur, K., 75
Matushima, J., 62, 65
McBurney, J. W., 53
McCaldin, R. O., 23, 42, 51
McCallan, W. E., 74
McCallum, A. W., 61, 62, 64
McCarroll, J. R., 119, 122, 125, 126
McCord, C. P., 5
McPhee, R. D., 23
Mead, J., 77, 78
Meeker, J. E., 19
Meneely, G. R., 78, 82
Menser, H. A., 66
Middleton, J. T., 61, 66
Middleton, W. E. K., 9
Moore, D. J., 19
Morgan, W. K. C., 139
Morgenson, A., 65
Mountain, I. M., 119, 125, 126
Mountain, J. D., 119, 125, 126
Murphy, E. M., 5
Murphy, S. D., 77

Nadel, J. A., 79, 81, 94
Nader, J. S., 9
Nakamura, K., 110
Navrotskii, V. K., 84
Negherbon, W. O., 73
Nelson, H. W., 91
Newberry, B. C., 22
Newill, V. A., 23
Newstein, H., 20
Niessen, H. G. L., 22
Noack, K., 63, 64
Norris, C. H., 22
Noyes, C. M., 7

O'Donoghue, J. G., 74
O'Gara, P. J., 63
O'Keeffe, A. E., 23
Olmstead, P., 75
Orning, A. A., 19
Ortman, G. C., 23
Oswald, N. C., 91

Pace, D. M., 84
Palmer, H. F., 22
Parcher, J. F., 7
Parker, A. 53, 54, 55
Pattle, R. E., 75, 76
Patty, F. A., 5
Perlman, R., 51
Pemberton, J., 128, 129, 136
Perry, W. H., 45
Petr, B., 142
Petrie, T. C., 54
Petrilli, R. L., 131
Phair, J. J., 91, 126, 139
Phillips, P. H., 91
Pitts, J. N., Jr., 8

Polezhaev, E. F., 98
Pollack, S. V., 126
Popov, I. N., 96
Prager, M. J., 8
Preston, R. St. J., 52
Prindle, R. A., 91, 130, 134
Prokhorov, Yu. D., 84
Pueschel, R. F., 8
Pushkina, N. N., 142

Ratner, I. M., 74
Reed, J. I., 125
Regan, C. J., 54
Rehme, K. A., 24
Reid, D. D., 128, 130, 132, 133, 137, 139
Reid, L., 74, 76
Renzetti, N. A., 7, 8
Rinehart, W. E., 106
Roberts, A., 91
Robinson, E., 9, 10, 12
Rodes, C. F., 22
Roesler, J. F., 9
Rogov, A. A., 84
Rohdin, J., 81
Rohrman, F. A., 19, 20
Ryan, T., 7
Ryazanov, V. A., 96, 97, 98

Salem, H., 78, 79, 94, 106
Saltzman, B. E., 21
Salvin, V. S., 55
Samorodiva, R. Ya. S., 23
Santner, J. T., 23
Sanyal, B., 51, 52
Scaringelli, F. P., 21, 24
Schikorr, G., 52
Schilling, F. J., 137
Schmidt, P., 142
Schnurer, L., 105, 108
Schueneman, J. J., 19
Schulz, R. Z., 75, 76
Schulze, F., 23
Schumsky, D. A., 126
Schwartz, C. H., 19
Scorer, R. S., 19
Scott, J. A., 120
Scott, W. E., 8
Selby, S. M., 10
Sellers, E. A., 74, 76
Seltser, R., 139
Semenenko, A. D., 98
Sereda, P. J., 51
Setterstrom, C., 62, 65, 66, 73, 74
Shaddick, C. W., 120, 128
Shashkov, V. S., 142
Sheffer, T. C., 61, 64, 65
Shekelle, R. B., 140
Shephard, R. J., 139
Shikiya, J. M., 24
Shock, J. M., 22
Shuck, E. A., 8
Sigmon, H., 75
Silverman, L., 94
Sim, V. M., 91, 95
Sjoholm, J., 81, 82
Skalpe, T. O., 143, 144
Smith, B. M., 7
Smith, W. S., 19
Solberg, R. A., 61, 66
Speizer, F. E., 28, 82, 83, 92, 93, 95
Spicer, W. S., Jr., 139
Spierings, F., 64
Sprague, H. A., 129
Spurr, G., 19
Stahl, R. W., 19
Stalker, W. W., 23
Stamler, J., 140
Standiford, N. E., 139
Steffens, C., 9
Sterling, T. D., 126
Stern, A. C., 8, 91
Stevens, E. R., 8
Stevens, R. K., 23
Stevens, W., 20
Stevenson, H. J. R., 9
Stokinger, H. E., 91
Stoklasa, J., 62
Stone, R. W., 133, 139
Storey, P. B., 139
Standberg, L. G., 77, 81, 82
Sullivan, J. L., 42, 65
Sussman V. H., 25, 52, 53
Swain, R. E., 62, 65

Tabor, E. C., 23, 27
Tamplin, B., 79, 94
Taylor, E. W., 139
Tendorf, R. B., 11
Terabe, M., 23
Thomas, A., 94, 110
Thomas, F. W., 8, 9
Thomas, M. D., 13, 25, 44, 45, 61, 62, 63, 64, 66, 76
Thompson, J. E., 24
Thompson, J. R., 84
Tice, E. H., 52
Tierney, D. F., 94
Tinker, C. M., 140
Tisdale, S. L., 45
Tomono, Y., 94
Toyama, T., 132, 139
Trakhtman, O. L., 96
Treon, J. F., 75
Trieff, N. M., 20
Turner, M. E., 139
Turner, T. H., 53
Tyler, R. G., 25

Underhill, D., 81, 107, 108, 109
Upham, J. B., 52, 55
Urone, P., 7

Van Den Heuvel, A. P., 7
Van Zuilen, D., 120, 122, 123, 125, 127, 129, 133, 136, 137, 142
Vassallo, C., 94, 110
Verma, M. P., 137
Vernon, W. H. J., 51, 52
Vintinner, F. J., 105
Von Lehmden, D. J., 19

Wagman, J., 7, 9
Wallace, A. S., 91
Waller, R. E., 11, 38, 54, 137
Walter, E. W., 119, 125, 126
Watanabe, H., 123, 126, 139
Weast, R. C., 10
Weaver, J. E., 61, 62, 65
Weedon, F. R., 73, 74
Wellington, J. R., 52
Wells, A. E., 61, 62, 63, 65, 66
Whitby, G. S., 61, 62
Whittenberger, J. L., 92, 93, 109, 110
Wicken, A. J., 128
Widdicombe, J. G., 79
Wilkins, E. T., 91, 120
Williams, J. D., 23
Wilson, R. H. L., 91
Winkelstein, W., 129
Witheridge, W. M., 5
Wohlers, H. C., 20
Wood, F. A., 61
Worcester, J., 92, 143, 144

Yanysheva, N. Ya., 142
Yocum, J. E., 51, 53
Yoder, R. E., 82
Yokiwa, Y., 79, 94
Yokoyama, E., 78, 82
Younker, W., 75

Zeidberg, L. D., 130, 134
Zickmantel, R., 134
Zimmer, C. E., 23, 33, 40
Zimmerman, P. W., 62, 66, 73
Zwi, S., 80

SUBJECT INDEX

A

Absorption
  of SO_2 in animals, 82–83
  nasal, 95
Acid titration method, 22, 23–24, 26–27
Acidity
  of rainfall and dustfall, 45
Age
  effect on animal exposure to H_2SO_4 mist, 75–76, 80–81
  effect on plant resistance to SO_2, 66
Air pollution episodes
  mortality figures during, 119–125
Air quality data
  SO_2, 33–47
Alfalfa
  effect of SO_2 on, 53–64, 67
Alpha rhythm, 96, 98
Aluminum
  effect of SO_x on, 52
Anemia
  Occurrence in highly polluted areas, 142
Asthma
  during air pollution episodes, 125
Atmospheric reactions of SO_x, 7–9
Australia
  concentrations of SO_2, 42
  effects of SO_2 on vegetation, 65
  morbidity studies, 132

B

Baltimore, Maryland
  morbidity study, 139
Barley
  effect of SO_2 on, 61
Blood formation
  effect of SO_2 on, 142
Blood pressure
  effect of SO_2 on, 92
Bronchitis
  correlation with SO_2 levels, 119–139, 140–142
  during air pollution episodes, 119–126
  mortality, 119–130
Bronchoconstriction
  effect of SO_2 on, 94
  mechanism in cats caused by SO_2, 79, 84, 85
Buffalo, New York
  mortality study, 129
Building materials
  effects of SO_x on, 54–56

C

CAMP (Continuous Air Monitoring Program) data, 33–47
Canada
  morbidity study, 134
Cancer
  mortality, 130
Catalytic oxidation of SO_2, 7
Chicago, Illinois
  effect of SO_2 on materials, 53–56
  morbidity study, 140, 141
Children
  epidemiologic studies of, 137–139
Chilliwack, British Columbia
  morbidity study, 134
Ciliary action
  effect of SO_2 on, 81–82
Citrus trees
  effect of SO_2 on, 62
Clinical visits during episodes, 125
Coal combustion gases
  mortality of animals from inhalation of, 105–106
Coal refuse burning
  emissions of SO_2 from, 19–20, 25
Coke processing
  emissions of SO_2 from, 20
Colorimetric (West-Gaeke) method, 21, 24–26, 33
Combined effects of SO_2 and H_2SO_4, 105–107
Combustion sources of SO_x, 5, 19–20, 25
Compliance (lung)
  effect of SO_2 on, 93
Conductometric methods, 21–22, 23–24, 25–26
Copper
  effect of SO_x on, 52
Corrosion of metals
  effect of SO_2 on, 51–53, 55–56
Corrosion rate, 51–53, 55–56
Cough reflex, 79–80
Coulometry, 23–24, 26
Czechoslovakia
  morbidity studies, 142

D

Detroit, Michigan
  mortality study, 123
Distribution of SO_2 in animals, 82–84
Dolomites
  effect of SO_2 on, 53–54, 56
Donora, Pennsylvania
  mortality study, 119
Dose-response curve, 83, 84–85, 110
Dublin, Ireland
  mortality study, 123
Dyed fabrics
  effect of sulfur compounds on, 55, 56

E

Economic Loss
  metal corrosion, 53
Electroconductivity
  measurement of SO_2, 33
England (see United Kingdom)
Epidemiologic studies, 119–142
Eston, England
  mortality study, 128, 129

F

Fading of fabrics
  effect of SO_2 on, 54, 56
Flame photometry measurement of sulfur compounds, 24, 26
Fog episodes
  mortality figures during, 119
Fuchsin-formaldehyde method, 23
Fumigation experiments, 63, 65

G

Genoa, Italy
  morbidity study, 131
Grasses
  effect of SO_2 on, 61, 62

H

Hospital admissions during episodes, 125
Humidity
  effect on irritant potency of H_2SO_4, 95
  effect on plant resistance to SO_2, 65

Humidity effect on atmospheric H_2SO_4 formation, 43–45
Hydrogen peroxide measurement method of SO_2, 21–22, 23, 26

I

Industrial exposure studies, 142–144
Irradiation of SO_2, 7

J

Japan
  mortality study, 123
  morbidity studies, 132, 139–140

K

Kerosene smoke, 106

L

Laboratory animals
  effect of SO_x on, 73–75, 76–80, 81–85
  effect of H_2SO_4 mist on, 75–76, 80–81, 84–85
  effect of zinc ammonium sulfate on, 81
Lead peroxide candles, 24–25, 26
Leaf tissue
  effect of SO_2 on, 61–65, 66–68
Leather
  effect of SO_2 up, 55, 56
Lifetime exposure of animals to SO_2, 82
Light intensity
  effect on plant resistance to SO_2, 66
Light scattering
  by H_2SO_4 particles, 9–10
  by sulfate particles, *9–10*
Limestone
  effect of SO_2 on, 53–54, 56
London, England
  mortality studies, 119–125
  morbidity studies, 125–126
Los Angeles, California
  morbidity study, 126
Lungs
  reaction to SO_2, 76–80
  reaction to H_2SO_4 mist, 80–81
Lung resistance
  effect of SO_2 on, 91–94
  effect of H_2SO_4 mist on, 94–95

M

Marble
  effect of SO_2 on, 56
Measurement methods
  of SO_2, 20–23, 24–25, 25–26
  of H_2SO_4, 24, 25, 26
  of sulfates, 24, 25, 26
Mechanism of SO_2 injury to vegetation, 63
Meuse Valley, Belgium
  mortality studies, 119, 120
Minute volume
  effect of H_2SO_4 mist on, 94–95
Monitoring programs, 33–47
Morbidity
  as an index of health, 117
  during air pollution episodes, 125–126
Morbidity studies, 130–142
Mortality
  index of health, 117
  due to combined effect of SO_2, H_2SO_4, and particles, 105–107
  due to SO_2 exposure, 73–75
Mortar
  effect of SO_2 on, 53–54, 56
Mucus
  effect of SO_2 on removal of, 95

N

Nasal resistance
  effect of SO_2 on, 94
Nashville, Tennessee
  mortality studies, 130
  morbidity studies, 134
National Air Surveillance Network (NASN), 33–47
New Hampshire (Berlin)
  morbidity studies, 134, 143
New York City
  mortality studies, 122, 123
  morbidity studies, 125–127, 137
Norway
  morbidity study, 143
Nutrient supply
  effect on plant resistance to SO_2, *66*

O

Occurrence of SO_x in the atmosphere, 5
Oil refineries
  morbidity studies in the vicinity of, 143
Optical chronaxie, 98–99
Oxidation of SO_2
  in power plant plumes, 8
  in nickel-smelting plumes, 8–9

P

Painted surfaces
  effect of SO_2 on drying time, 51, 56
  effect of SO_2 on glossiness, 51, 56
  general effect of SO_2 on, 51, 56
Paper
  effect of SO_2 on, 55, 56
Particle size
  effects on animal response to pollutants, 79–81
  distribution of sulfates, 9
Pennsylvania (Seward-New Florence)
  morbidity study, 134
Persia (southern)
  morbidity study, 143
Petroleum refineries
  emissions of SO_2 from the vicinity of, 19–20, 25, 42, 47
pH (see acidity)
Photochemical oxidation of SO_2, 7–8
Photooxidation rate of SO_2 7
Pine trees
  effect of SO_2 on, 61, 62, 64–65, 67–68
Pittsburgh, Pennsylvania
  effect of SO_2 on materials, 52
Plants
  effect of SO_2 on, 61–65, 66, 68
  effect of H_2SO_4 mist on, 67–68
Pneumonia death rate, 128
Potentiation of SO_2, 108, 109
Power plants
  SO_2 in the vicinity of, 42, 47
Pulmonary function
  as an index of health, 117–118
  effect of simultaneous exposure to SO_2 and NaCl, 108, 110
  effect of simultaneous exposure to SO_2 and H_2SO_4, 107–108
  effect of SO_2 and H_2SO_4 mist on, 91–95
Pulp mills
  morbidity studies in the vicinity of, 143
Pulse rate
  effect of SO_2 exposure on, 92

R

Refuse Incineration
  emissions of SO_2 from, 19–20, 25
Respiratory rate
  effect of SO_2 exposure on, 92
  effect of H_2SO_4 mist on, 94–95
Respiratory response, 76–79
Rotterdam, The Netherlands
  mortality studies, 123
  morbidity studies, 127, 137

S

St. Louis, Missouri
  effect of SO_2 on materials, 52–54
Salford, England
  mortality study, 129
  morbidity study, 136

Sampling techniques for SO_2, 20–27
Slate
  effect of SO_2 on, 56
Smelters
  emissions of SO_2 from the vicinity of, 19–20, 25, 42
Soil moisture
  effect on plant resistance to SO_2, 66
Soiling index relationship to morbidity, 145
Sources of atmospheric SO_x, 19–20, 25
Spectroscopic measurement methods of SO_2, 22, 24, 26
Steel
  effect of SO_x on, 51–54, 55–56
Sulfation plate, 24
Sulfation rates, 20–27, 55–56, 144–147
Sulfur oxides
  effect of sunlight on, 9–14
Sulfuric acid
  relationship to SO_2, 42–45
  formation during photo chemical reactions, 7
  emissions of SO_2
    manufacture of, 19–20, 25
Sulfuric acid mist
  effect on laboratory animals, 75–76, 80–81
  effect on man, 94–95
  effect on materials, 55–56
  effect on plants, 66, 68
  measurements of, 25, 27
Susceptibility of vegetation to SO_2, 61–68
"Susceptible population," 119
Suspended sulfates
  concentrations, distribution, and measurement of, 25, 26
Synergism of SO_2 and NaCl, 111
Synergistic effects
  SO_2 and O_3 effects on tobacco, 66
  NO_2 and SO_2 effects on tobacco, 66

T

Temperature
  effect on plant resistance to SO_2, 65
Tennessee (eastern)
  effect of SO_2 on vegetation, 64
Textiles
  effect of sulfur compounds on, 55–56
Threshold concentrations of sulfur compounds brain sensitivity, 96, 98, 99
  eye sensitivity, 96, 97, 98–99
  odor perception, 96–97, 99
Tidal volume
  effect on H_2SO_4 mist on, 94
Tobacco plants
  effects of SO_2 and O_3 on, 66
  effects of NO_2 and SO_2 on, 66
Trail, British Columbia
  concentrations of SO_2, 42
  effects of SO_2 on vegetation, 64
Trees (see also Pine and Citrus trees)
  susceptibility to SO_2, 63–65

U

U.S.S.R.
  morbidity studies, 138, 139, 142
United Kingdom
  mortality studies, 127, 128
  morbidity studies, 136, 137, 138

V

Visibility reduction
  by SO_x and H_2SO_4 mist, 9–14

Vital capacity
  effect of SO_2 on, 91, 99

W

West-Gaeke method (see also Colorimetric method), 21, 22, 24–25, 25–26
Wind speed effect on atmospheric H_2SO_4 formation, 43

Z

Zinc
  effect of SO_2 on, 52
Zinc ammonium sulfate
  effect on laboratory animals, 81

ACKNOWLEDGEMENTS

The following sources, in most instances the copyright holders, have granted permission to the National Air Pollution Control Administration to include the following figures and tables in Air Quality Criteria for Sulfur Oxides:

Table 1-2.—McGraw Hill, Inc., New York *and* Chemical Rubber Company

Page 20—American Institute of Chemical Engineering, New York; *and* Electrical World Copyright 1967, all rights reserved by McGraw Hill, Inc.

Page 42—National Research Council Ottawa, Canada

Table 3-4.—Pergamon Press, Inc., New York City

Table 9-1.—Research Institute for Public Health Engineering, Delft, The Netherlands

Figure 5-1.—American Society of Plant Physiologists, Washington, D. C.

Figure 9-1.—Royal Society of Health

Figure 9-2.—Royal Dublin Society, Dublin, Ireland

Figure 9-3.—American Medical Association, Chicago, Ill.

Figure 9-4.—Lea and Febiger, Philadelphia, Pa.

Figure 9-5.—British Medical Journal, London

Figure 9-6.—Air Pollution Control Association, Pittsburgh, Pa.

☆ U. S. GOVERNMENT PRINTING OFFICE : 1969—347-411/31